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KERATOMALACIA.*

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FEW diseases of the eye present a more characteristic appearance than that known as "keratomalacia."

The disease is tolerably common in some parts of the world. In Russia it is even frequent towards the end of the great Lenten Fast and after the fortnight's fast in August. The mothers lose their milk, and in consequence the babies' eyes suffer. It is also common among the half-starved negro children in South America, where it is called "ophthalmia Braziliana," a name given to it by Gama Lobo.(1) According to Kollock (2) the malady is common among the young negro children of Charleston, the largest city of South Carolina, where more than one half of the inhabitants are coloured. The disease was never seen in the whites. In Kollock's experience, when the cornea in these cases suppurated, treatment availed little, and the patient soon died. Herbert (3) and Yarr (4) tell us that the disease is common in the native quarter of such great towns as Bombay, Calcutta, and Hong Kong. Lastly, in Japan this peculiar disease of children is well known, where it goes

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by the name of "Hikan." It appears epidemically in that country during the months July to September, when diarrhœa is rife (Mori [5]).

A similar condition is found, although it is not common, in England, exclusively in babies whose nutrition is at a dangerously low ebb. The disease was described in 1827 by Dr. Joseph Brown, of Sunderland ('Edin. Journ. of Med. Science,' vol. iii, p. 218). It is probably more familiar to physicians attached to children's hospitals than to ophthalmic surgeons, for the subjects of keratomalacia are often dangerously ill, and on that account are more likely to be taken for advice to the former institutions. A few cases, however, have been published in this country by Sir William Bowman, Holmes Spicer, Tatham Thompson, A. W. Ormond, and myself.

From the figures at my disposal (6) I estimate that keratomalacia is encountered in about 1 per 1000 of the eye cases attending a children's hospital in the poorer part of London.

It is essentially a disease of early infancy. The average of my own thirty-one cases was 4·54 months.

I have never seen the disease except in the children of the poor.

Any disproportion between the sexes, as shown by the figures given by Knaebel (7), Thalberg (8), and Braunschweig (9), is almost certainly fortuitous. Of my own cases, thirty-one in number, 41 per cent. were in males, 54 per cent. in females, and in 3 per cent. the sex was not noted.

Von Graefe (10), who was familiar with the disease in Berlin, attributed keratomalacia in infants to encephalitis. Indeed, the changes believed to be those of encephalitis were found by Prof. Klebs in two of von Graefe's patients, aged 3 and 4 months respectively. This view was adopted by other ophthalmologists, notably by Hirschberg (11). But further pathological work proved that the so-called "diffuse infantile encephalitis" of Virchow was non-existent, and that the granulo-fatty corpuscles, once regarded as evidences of encephalitis, can be found in the brain of all little children (Jastrowitz [12). The encephalitic theory, therefore, was generally abandoned.

Before long another hypothesis occupied the ground, namely, that keratomalacia was the outward and visible sign of a generalised bacterial invasion. Some of the cases which were reported appeared to support this view. One of the most important of these was by Leber and Wagenmann (13). A male baby, aged 10 days, manifested necrosis of the ocular conjunctiva around the cornea of one eye. He

died a few hours after he was first seen. The autopsy failed to reveal any cause adequate for death, and, in especial, there was no evidence of encephalitis. Bacteriological investigation, however, brought to light the fact that a multiple streptococcal invasion existed. Streptococcal chains were found in the vessels of the tissues of the eye and in the other organs examined, which included the kidneys, the suprarenal bodies, and the skin. By Leber and Wagenmann this bacterial invasion was believed to be responsible both for the ocular affection and for the lethal issue of the case. In this connection a case by J. E. Weeks (14), of New York, may be quoted : An emaciated and bottle-fed baby, aged 8 months, suffered from xerosis of the conjunctiva, along with ulceration of the corneæ. Death from exhaustion. The autopsy showed a fatty liver and enlarged spleen and kidneys. The pelvis of the right kidney was filled with flocculent pus, while its lining mucous membrane was coated with a frothy and whitish exudate, resembling to some extent the secretion observed on the conjunctiva during life. The xerosis bacillus was present in conjunctiva, pelvis of the kidney, liver, spleen, and pancreas.

The theory of endogenous infection, as implied by the foregoing cases, may, in my opinion, be put aside without hesitation. The experiences of Baumgarten and of Braunschweig prove that, as a rule, micro-organisms can be grown neither from the blood during life nor from the viscera after death of children affected with keratomalacia. The whole of the available evidence goes to show that the determining cause of the corneal ulceration is to be found in exogenous infection with this or that pyogenic micro-organism, while its predisposing cause is the lowered vitality of the patient.

On a general inquiry into the causes of keratomalacia we are at once struck by the fact that the disease hardly ever occurs in breast-fed babies. Of Knaebel's eighteen cases three only, or 16·6 per cent., had been nursed by their mothers. Of my own cases, again, two alone, or 6·46 per cent., were entirely breast-fed.

Without exception the victims of this pitiful disease suffer from marasmus, either of a symptomatic or idiopathic character. The causes of the former include tuberculosis, syphilis, and chronic diarrhœa and vomiting, the familiar "D. and V." of a children's hospital. The name "marasmus" or "athrepsia," however, may be more properly applied to the idiopathic group, where, as well known, neither examination during life nor dissection after death enables us to ascertain the cause of the malassimilation.

In London there is a distinct connection between keratomalacia, on the one hand, and the disease variously known as "epidemic

enteritis," "zymotic enteritis," "ileo-colitis," or "epidemic diarrhœa," on the other. By contrasting the seasonal curves of the two ailments it can be shown that June, July, August and September represent the greatest mortality from "epidemic diarrhœa" and the greatest incidence of keratomalacia. Indeed, in the metropolis keratomalacia has a distinct seasonal incidence. This is well borne out by the fact that about three quarters of my cases have been met with in the six warm months (May to October) and only about one quarter during the six colder months (November to April).

Although "epidemic diarrhœa" is the outstanding cause, yet it must not be forgotten that there are others, of which the most important are—(1) syphilis, (2) tuberculosis, and (3) marasmus, pure and simple. A few words may be usefully devoted to the discussion of each of these in turn.

The syphilitic origin of keratomalacia has been insisted on by several writers, especially by Horner (1882), Biber (1890), Zirm (1895), Peltesohn (1898), and Terrien (1905). Horner's testimony (15) is particularly interesting. He met with cases of keratomalacia which recovered and which later developed true parenchymatous keratitis, and he also saw in the same family an infant succumb to keratomalacia and athrepsia, while a brother presented the classical signs of Hutchinson's interstitial keratitis. Wicherkiewicz (17), indeed, if I understand him aright, denies the ability of any factor other than hereditary syphilis to cause the disease. The discovery by myself (18) of the *Spirochæta pallida* in scrapings from the diseased cornea constitutes the scientific proof of the reality of syphilis as a factor in the causation of some cases of keratomalacia.

Of my thirty-one cases of keratomalacia, syphilis was diagnosed clinically in seven, or in 22·58 per cent. The Wassermann reaction was not available when most of my figures were collected.

My figures are small, but so far as they go they indicate that tuberculosis is an important cause of keratomalacia in infants. It existed in six, or 19·36 per cent. of my cases, the diagnosis being made during life in three and after death in three of the children. It is to be noted in passing that of the twelve fatal cases tubercle was present in one quarter. The disease was of the generalised type, as is usual in children of so tender an age, with pulmonary or meningeal manifestations.

Marasmus, pure and simple, accounted for seven, or 22·58 per cent. of my thirty-one cases. It is of practical importance to remember that at such an early age there are many points of

resemblance between marasmus on the one hand and tuberculosis on the other. Progressive wasting, pallor, and loss of strength are features shared by both affections. Many a case assumed to be one of marasmus has been found upon the dissecting-room table to be one of tuberculosis.

Finally, in two of the cases, or 6·45 per cent., the cause of the corneal mischief remained altogether obscure.

When all is said and done, we shall not be far wrong if we assume that destruction of the cornea may supervene, other things being equal, in any baby whose nutrition is seriously impaired, whether as the result of disease, semi-starvation, or, for that matter, from any cause whatsoever.

That the condition responsible for keratomalacia often ends fatally is, of course, unquestioned. It is, however, not correct to speak, as some writers have done, as if a lethal termination was inevitable. A glance at the figures to be found in the literature will prove this point. Of Knaebel's eighteen cases, exactly one half died before they reached the age of six months. In my own series of thirty-one cases the mortality amounted to thirteen, or 41·93 per cent. This number is probably below the mark, since some of my patients may have died soon after they passed from under my personal observation. The factors that have probably most to do with the issue, lethal or otherwise, are: (1) The age of the child—the older the patient the better is his chance of recovery; and (2) the nature of the underlying disorder, since, in my experience, syphilitic and marantic cases justify a better prognosis than those associated with tuberculosis.

Everybody knows that babies affected with keratomalacia are apt to die from broncho-pneumonia. That condition was found, either during life or after death, in more than one third of my fatal cases, the exact proportion being 38·46 per cent. In some instances the pulmonary condition is in the nature of hypostatic pneumonia, which is frequently the cause of death in cases of marasmus. In another and perhaps a larger contingent, however, I cannot resist a suspicion that the broncho-pneumonia merely represents the outstanding symptom of a generalised tuberculosis which has escaped (as it may readily do) recognition.

The clinical features of a marked case of keratomalacia may be described as follows:

The baby, who is obviously wasted and very ill, lies placidly in his mother's arms, and although he may whine at intervals, yet he shows few signs that he is suffering from a severe and destructive disease of the eyes. He may present the "old-mannish" look so

characteristic of advanced cases of marasmus. His skin is lax and withered, so that when pinched up between the fingers it retains for some time its unaccustomed form. He is white. His temperature is subnormal. There is usually a tendency to diarrhœa and vomiting.

The eyelids, soft and supple, are not reddened or swollen. They can be opened with scarcely any protest on the part of the baby. Few things, indeed, are more striking in keratomalacia than the paucity of external inflammatory changes. On separating the eyelids, however, the gravity of the condition is at once apparent. One or usually both corneæ may be involved in an ashen-hued slough, through which the iris may be seen to have prolapsed here and there. But there is neither lacrimation, chemosis, nor congestion of the eyeball—in a word, none of the appearances that we should naturally expect to find associated with sloughing cornea. To employ the graphic words of J. N. Fischer (19) (one of the earlier writers on the disease), the eyes "have a corpse-like appearance in the still living body."

One peculiar feature (first described by von Graefe), namely, xerosis, calls for a word of separate mention, although it is not essential to the diagnosis of keratomalacia. It was noted in 41·93 per cent. of my own cases. When present in little children it is almost pathognomonic of keratomalacia. This change usually takes the form of a small, triangular, greasy-looking white patch situated in the ocular conjunctiva on the inner or the outer side of the cornea. In several of my own cases, however, it lay below the cornea, and I have more than once seen it encroach upon the cornea itself. Sometimes the cornea, or a considerable part of its circumference, is encircled by a narrow, slightly raised, glistening white line of xerosis occupying the position of the limbus, the conjunctiva in the neighbourhood of which is often lustreless, dry, and thrown into wrinkles. Once alone have I seen the xerotic changes affect the palpebral conjunctiva.

The changes of xerosis are apparently due to the lodgment and multiplication *in situ* of the saprophytic xerosis bacillus, together with the local precipitation of the Meibomian secretion (Reymond).

The eyes often look dry, and are more or less insensitive to stimuli, and in severe cases a characteristic feature is that the baby scarcely resents attempts to examine his eyes, or, at most, moans feebly and piteously during the necessary manipulations. This alone should arouse suspicion as to the nature of the case, suggest extreme care in handling the eyes, and, lastly, prompt a bad prognosis.

If the baby live long enough symptoms of panophthalmitis may supervene, when the entire clinical picture undergoes a change. For example, the lids become red and swollen, there is discharge from the eye, the xerotic patches (if they were present) disappear, the eyeball becomes violently inflamed, and, indeed, the signs of reactionary inflammation become so marked as almost to arouse a suspicion that a gonorrhœal infection has been superadded to the original mischief. Thalberg, speaking of this class of case, states that twice he has reached the mistaken diagnosis of blennorrhœa with secondary gangrene of the cornea.

Under any circumstances, the outcome of the case is only too likely to be unfavourable, for even if the baby survive he will probably be left blind of one or both eyes. The destructive nature of the disease is shown by figures recently published by A. Schiele (20), dealing with thirty-three cases of keratomalacia seen in the Kursk district of Russia in the years 1904, 1905, and 1906. Thirteen of the children were rendered totally blind, and seven were blinded in one eye; two recovered with corneæ blemished, and eleven could not be traced. Again, of the nine children mentioned in Knaebel's series who survived, eleven of the thirteen eyes affected (*i. e.* five unilateral and four bilateral cases) were known to have been blinded by the disease.

The final results of the disease may be phthisis bulbi, staphyloma, keratectasia, or a more or less serious leucoma adherens.

The diagnosis of keratomalacia should offer no difficulty to anybody who is aware of the existence of the disease. It is important to bear in mind that neither the existence of xerosis nor the presence of corneal changes in both eyes is essential to the recognition of the malady. It would be a fair generalisation to say that ulcers of the cornea, especially if bilateral, in infants between the ages of two and twelve months, are far more likely to be due to keratomalacia than to any other cause. Such children are too old for the corneal complications of gonorrhœal ophthalmia, and almost too young to suffer from the other great cause of corneal ulceration in children, namely, eczematous keratitis. While this is true, it is rather curious to notice from the case reports scattered throughout literature how often the symptoms of keratomalacia have been confused with those of ophthalmia neonatorum, although the age-incidence of the two ailments is very different. Indeed, sometimes the distinction between the diseases is not altogether simple, a fact of which I have had personal experience.

Keratomalacia may be said to be a malady devoid of distinctive

pathology. Considerable importance has been attached by Ernst and some others to the deposition of keratohyalin in the superficial layers of the conjunctival epithelium. The coarse pathology of any given case, however, will depend essentially upon the stage when the eye is examined.

If the pathology be not distinctive, neither is the bacteriology. No causal micro-organism has yet been discovered. Xerosis bacilli, staphylococci, streptococci, pneumobacilli, *coli* bacilli, gonococci, and, in particular, pneumococci have been found in scrapings from the sloughing corneæ. The diversity and multiplicity of these results is suggestive of there being no micro-organism peculiar to keratomalacia. As to my personal investigations, the only point worth noting is that the pneumococcus was present in five (45·45 per cent.) of eleven of my cases, twice in pure culture and thrice mixed with other microbes. The pneumococcus has been found in keratomalacia by other observers besides myself, as E. v. Hippel (21), Knaebel (7), Braunschweig (9), and Dötsch (22). That it is not essential to the suppurative process is shown by the fact that it need not necessarily be present in cases of keratomalacia. Before leaving this part of the subject I may say that three of my cases go to show that, on rare occasions, the gonococcus may affect the cornea and produce sloughing to the exclusion, complete or almost complete, of the conjunctiva.

Almost everybody, from Dr. Joseph Brown downwards, who has written about keratomalacia has compared the process, aptly enough, with that brought about in dogs fed, as in Magendie's famous experiment, with sugar and distilled water or other non-azotised food. In those animals an early and constant sign of inanition, as everybody knows, was furnished by sloughing of the corneæ and consequent destruction of the eyeballs. Indeed, in the light of modern knowledge it cannot be doubted that keratomalacia is due remotely to lowered vitality in a tissue devoid of blood-vessels, and immediately to inoculation with this or that pyogenic microbe. The assumption of a causal micro-organism is quite unnecessary.

Amongst exciting causes stress has been laid by various writers upon slight injuries to the epithelium (Stephenson), exposure by inadequate closure of the eyelids (Horner, Zirm), and even to pressure of the eyelid upon the tissues of an enfeebled cornea (Förster, Thalberg).

The corneal changes, in fact, appear to be analogous to the localised gangrene of the skin over the sacrum, the heels, and other bony prominences which is sometimes observed towards the terminal stages of marasmus, symptomatic or idiopathic. It is of interest

to note that such acute bed-sores (for that is what they are) have been noted in cases of keratomalacia by myself and other observers.

Treatment.

The general treatment, speaking broadly, is that suitable for marasmus—as, for example, hospital surroundings, a plentiful supply of fresh air, an incubator, a wet nurse, suitable food, and the administration of small doses of alcohol. Cod-liver oil has a distinct value in these cases provided it can be retained by the patient. Mori believes that keratomalacia is essentially due to a deficiency of fat. On that view, in spite of the diarrhœa often present, he has used cod-liver oil, and by that means has succeeded in curing even severe cases of the Japanese "Hikan." In my experience, however, if the oil causes diarrhœa, it is best given by inunction. Neats-foot oil is an old-fashioned but efficacious remedy in these cases. The fact is significant that some of the cases of keratomalacia that have recovered have done so under specific treatment. The rapidity of action desirable in these desperate cases is probably best secured by giving mercury with chalk in doses of from ½ gr. to 1 gr. three times a day, and by rubbing into the skin every night a piece of mercurial ointment, roughly of the size of a pea. In order to prevent the medicine from being carried away by the bowels, it is advisable to combine with each dose of the mercurial ½ gr. of sodium bicarbonate and 1 gr. of aromatic powder of chalk.

The local treatment should include the frequent use of hot saline or boric lotion. The fluid should be directed against the closed eyelids for ten or fifteen minutes at a time, once a day or oftener, and in the intervals between the applications the lids should he kept tied up by means of a bandage. Gama Lobo and de Gouvêa speak highly of steam at a temperature of 40° C. employed in this way. I can testify that it is sometimes of great service. The heat both limits necrosis and hastens separation of any sloughs that may be present. At intervals an antiseptic, such as argyrol, quinine, or hydrogen peroxide, should be dropped into the eye. My own experience leads me to rank physostigmine highly in keratomalacia. The alkaloid should be dissolved in sterile oil (½ per cent. to 1 per cent.), and the collyrium be dropped into the eyes two to six times a day. Physostigmine was strongly recommended by J. Thalberg, who saw much of the disease in Russia, and its use has been endorsed by Holmes Spicer (23) and by Adolf Knaebel (7).

It is sometimes surprising to see how quickly the cornea casts its slough under the use of the remedy.

In the more acute stages of the disease the utmost gentleness should be exercised by making the necessary examinations and applications. The importance of care in this respect is shown by cases reported by Thalberg and Biber respectively, where the lens escaped from the eyes of babies on attempting to separate the eyelids. A precisely similar accident happened in one of my own cases.

Speaking from the standpoint of my present experience, I should now be chary of applying any strong antiseptic, as carbolic acid, to these ulcers, while I certainly should not attempt to scrape or cauterise them. The vital powers appear to be so low that the cornea is unable to furnish the reaction necessary to healing. The only operation that I might be tempted to perform would be the Guthrie-Saemisch section, or, possibly, the multiple peripheral punctures of the cornea described many years ago by the late George E. Walker, of Liverpool, under the name of "kerotomy" (24).

The last point is that if the baby's eyelids are incompletely closed it may be advisable either to suture them together or else to perform tarsorrhaphy.

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THE SURGICAL ASPECTS OF ACUTE ABDOMINAL DISEASE IN CHILDHOOD.*

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The varieties of abdominal emergencies which occur in childhood are in a measure similar to those of adult life. When confronted with a case in childhood presenting acute peritoneal symptoms we are probably justified in excluding an origin in the gall-bladder, the pancreas, and in the diverticula of the colon. When the symptoms point to intestinal obstruction we may with some justification dismiss several varieties, common in adult life, from our consideration. For instance, internal herniæ and gall-stone obstruction practically never occur in childhood; neoplasms in the wall of the bowel hardly require consideration, although I would remind you that carcinoma of the bowel has been recorded in early life as has also the rare intestinal sarcoma.

My own experience of 98 cases of the "acute abdomen" in children under twelve years of age shows the diseases represented as follows:

Appendicitis	54 cases.
Pneumococcal peritonitis	6 „
Inflammation of the diverticulum of Meckel	2 „
Intussusception	28 „
Intestinal obstruction by a band	3 „
Intestinal obstruction from adhesions	4 „
Intestinal obstruction from pressure of mesenteric glands	1 case.

In the above list there is no mention of gastric or duodenal ulcer, since no such case has come under my observation. It is usually stated that gastric ulcer is a rare disease in childhood, but the recent literature seems to suggest that it is by no means so rare as

* A paper read before the Richmond Division of the British Medical Association on April the 26th, 1911.

is usually supposed. The acute perforation of the ulcer is, however, probably decidedly rare. Nevertheless it has been recorded at all ages, even shortly after birth. In infants and young children it is stated that there have not been any symptoms preceding the acute catastrophe by means of which a diagnosis might have been made. In older children, as in adults, symptoms pointing to gastric trouble have been present. The possibility of a perforated gastric ulcer should always be considered in the differential diagnosis of any anomalous case of acute peritonitis in childhood. I would remind you that gonorrhœal peritonitis may occur in childhood, that typhoidal and tuberculous ulcers may perforate, and that the pedicle of an ovarian tumour may undergo torsion with acute symptoms. Of rare varieties of intestinal obstruction may be mentioned foreign bodies, enteroliths and worms obstructing the lumen of the canal, and also the final acute phase of a slowly produced obstruction by a congenital stricture, or that interesting disease, the so-called congenital idiopathic dilatation of the colon. All such conditions are infrequent, and time permits me merely to mention them. It will be much more profitable to consider the more common varieties of acute abdominal disease in children which we meet with in every-day practice.

Appendicitis.

Appendicitis is undoubtedly the most common acute abdominal disease in childhood requiring surgical treatment. It is a disease which is so familiar to all that some apology is due to you for bringing forward such a hackneyed subject. My plea must be that appendicitis still has a high mortality in childhood, and for this we are to a certain extent to blame, and a disease so frequently discussed as appendicitis cannot be without interest, for there cannot be unanimity of opinion on all points. I have no intention of giving a detailed account of appendicitis, but merely wish to make a few remarks upon the disease in childhood and the opinions at which I have arrived.

The word "appendicitis" requires qualification, for, as you know, the disease may be caused by the tubercle bacillus, the pneumococcus, actinomycosis (doubtful in childhood), as well as by the bacillus coli and the common pyogenic cocci. Of the former varieties I shall say nothing, but confine my remarks to the common variety which is produced by the colon bacillus or the common pyogenic cocci.

Appendicitis is a chronic disease with acute exacerbations. The

surgeon naturally sees it in its acute phase. Only in five out of fifty-four cases in children have I operated to remove an appendix in what is called the "quiescent interval." Appendicectomy during the "quiescent interval" is perhaps the most common operation in adults. In children, on the other hand, operation is generally undertaken for suppuration. In no less than thirty-nine cases out of fifty-four I have operated for peritonitis, diffuse (eighteen cases) or localised (twenty-one cases). In ten further cases I was fortunate enough to be able to operate within the very early hours of the acute illness where symptoms may be said to have been comparatively trivial, and whether resolution or suppuration would have occurred in such cases cannot be said. These figures, anyhow, will show that appendicitis in childhood comes generally to the surgeon on account of suppuration. Hence it is considered that appendicitis is a more severe and virulent disease in children and claims a relatively higher death-rate than in adult life. It is also stated that appendicitis is comparatively infrequent in the first decade of life. Statistics usually give the common age as fifteen to twenty-five years or thereabouts. The evidence of chronicity of the disease in the child is shown by the state of the appendix removed during the acute phase of the illness. In all my cases of diffuse peritonitis I have removed the appendix. In the earlier ones of localised suppuration I was content with merely opening the abscess, but in the later ones (the majority) I have almost invariably removed the appendix in addition, and have never had any cause to regret so doing. The pathological changes in the appendix in the diffuse and the local suppuration are similar. Whether the inflammation be localised or diffused is merely a battle between the virulence of the organisms and the resisting powers of the individual. The appendix occupies a variable position, and it might be thought that one tucked away behind the cæcum or colon would be less liable to infect the general peritoneal cavity than one lying internal to the cæcum directed towards the pelvis or even hanging into the pelvic cavity. Such is not the case, however, for the posteriorly or externally situated appendix is relatively just as commonly the cause of a diffuse peritonitis as is the appendix which occupies the more frequent internal position. The appendices which I have removed during the acute phase of the disease almost invariably showed changes of some duration. In at least three quarters of them one or more concretions were found; in many a naked-eye examination of the walls showed obvious chronic changes, and in others a microscopical examination proved old-standing disease.

In many of these cases the previous history relates symptoms which undoubtedly pointed to appendicular disease. In my experience the most frequent history is that the child, otherwise perfectly healthy, has suffered from recurrent stomach-aches, usually associated with vomiting, perhaps diarrhœa, and if the temperature be taken, a little fever. The illness is so comparatively trivial in the parents' judgment that medical advice is not often asked. When the appendix is removed, these attacks no longer occur. I am not contending that all stomach-aches in children owe their origin to the appendix, but there is no doubt whatever that many of them do. You must remember that the child will rarely locate the pain to the appendix region, but will pass his hand right across the abdomen when asked where the pain is. But if an opportunity arises of examining a child who develops these symptoms abruptly, and in addition definite tenderness and muscular rigidity, however slight, is present in the right iliac fossa, the right loin, or in the hypogastric regions, then appendicitis can be pretty certainly diagnosed. In other cases it was stated that the child was constantly ailing, the bowels were irregular, sometimes there was constipation and sometimes diarrhœa, there were periods of anorexia, occasional febrile intervals associated with colic and abdominal discomfort. Here, again, these symptoms are not always diagnostic of appendicitis, but localised tenderness over the lower right abdomen would strongly suggest the appendix is at fault. It is very noteworthy that when the appendix has been removed in such cases the improvement in the child's health is very marked. In a certain number of cases the previous history is absolutely negative, and this in spite of the fact that the appendix shows changes of some duration. The more carefully the previous history is inquired into the fewer will be the number of cases in which it is absolutely negative. It can easily be understood how previous symptoms may have been overlooked, for parents cannot recall with any degree of accuracy the various abdominal ailments from which their children have suffered. The symptoms are frequently so slight that they pass unknown, or are altogether ignored and forgotten.

An inquiry into the previous history of adolescents who come for operation for appendicitis often reveals the fact that the disease may be traced back to childhood. In all probability appendicitis is extremely common in the first decade of life, but at that period comes under the notice of the surgeon much less frequently than in the second decade.

Time does not permit me to enter at all fully into the *diagnosis*

of appendicitis, and I must rest content with merely calling to your notice a few points. The signs and symptoms are well known, and do not differ, save in degree, in any form of the disease. I wish to lay especial stress upon the recognition of the minor manifestations of the disease. I know perfectly well that probably in the majority of cases medical advice is not sought at this period of the disease. In many cases, therefore, we cannot hope to save the children from the perils of a grave appendicitis until we can educate the parents to regard all stomach-aches in their children as serious and at once to seek advice. We on our part must never disregard any stomach-ache or abdominal discomfort in the child until we have thoroughly satisfied ourselves of its nature. I would repeat that many of these stomach-aches in children, regarded as quite trivial and almost as a part of the child's life, owe their origin to the appendix. An abrupt onset of abdominal pain (the child will never localise it to the right iliac fossa, but draw the hand all over the abdomen when asked where the pain is), usually with vomiting, perhaps diarrhœa, and fever, however slight, associated with localised tenderness and muscular rigidity, however slight, means almost certainly appendicitis. It is only the association of these signs which can establish the diagnosis. The illness may be over in the space of a few hours—indeed, anything like severe pain may only last an hour or so—but for a few hours afterwards some tenderness can be obtained on deep pressure on the abdominal wall. If parents do not consult their medical practitioner for these symptoms only rarely will the surgeon have the opportunity of seeing the child in one of these minor manifestations of the disease. Hence it has been my lot to have had the opportunity to operate on 10 occasions only in such cases. The symptoms appeared at first sight to be quite trivial—indeed, in some, spontaneous pain had practically disappeared, but localised tenderness and slight rigidity of the abdominal muscles persisted. It is interesting to note the condition of the appendix in these cases. The adhesions were slight or absent, the appendix was inflamed in varying degree and for a variable extent, a concretion may be present in it, and, what is most important even at this early stage of the acute infection, a limited infective gangrene may be present, and this gangrenous process may extend practically throughout the entire thickness of the wall of the appendix. However slight may be the symptoms in the child the condition of the appendix cannot be foretold.

A very common symptom during the acute phase of the disease in children is frequency of micturition, and perhaps some pain during

the act. This is so commonly present that I always ask directly for it.

I admit that only rarely have we an opportunity of seeing a case presenting such minor symptoms, but such is the beginning of many of the graver forms of appendicitis in children, and it is only because the child does not get better that after twenty-four, forty-eight hours or longer medical advice is sought. At this period there might be difficulty in the differential diagnosis, and also in the degree of appendicitis.

A severe peritoneal infection is occasionally associated with cerebral symptoms, and these may appear at first sight to be the predominating features of the disease. It is by no means surprising that cerebral symptoms may occur in acute infective conditions if we remember the changes which Crile has described in the cells of the brain in acute toxic conditions (and other conditions also). Similar cerebral symptoms will occasionally occur after a severe operation upon a child.

More frequently than the latter an acute peritoneal lesion may be confused with an acute pulmonary affection. A child with pneumonia may complain severely of abdominal pain, the abdomen may be distended and tender, and there may be a certain degree of rigidity of the abdominal muscles. The aspect of the child, the pulse-respiration ratio, and the absence of true muscular rigidity, as shown by the relaxation of the abdominal wall during the height of inspiration, even in the absence of physical signs in the chest, will generally serve to differentiate the two conditions.

In a few cases with ultra-acute symptoms the clinical picture of diffuse peritonitis, as described in the text-books, is obvious in a few hours after the onset of the illness. This, however, is not common in the child. More frequently diffuse peritonitis is insidious in its onset and spread. The abdominal wall may move freely on respiration, and the child may be able to raise his shoulders from the bed with comparatively little discomfort. To the touch the abdominal wall is not hard—indeed, to light palpation it may be quite soft. There is neither abdominal distension nor retraction. The child may sleep for hours. The temperature may be normal or very slightly raised. The pulse-frequency, always variable in the child, may be below 100 if taken asleep. There is always, however, to be detected some spasm of the abdominal muscles upon the pressure of the hand or finger-tips, and in addition the aspect of the child is suggestive of acute toxæmia. There should be no doubt about the diagnosis of this type of diffuse peritonitis.

In some cases after a variable interval of the minor manifestations of the disease a sudden and very severe pain is experienced. The symptoms are comparable to the perforation of any hollow viscus, and always denote a perforation of the appendix. There should be no delay when once this has occurred. If there be, the course taken will be similar to that of a perforation of any other viscus. Within a few hours the pain abates, and the condition seems to be improving, but with certainty it may be said that diffuse peritonitis or localised suppuration will occur: which of the two it cannot be foretold.

Not infrequently after a typical onset a sudden cessation of pain occurs. I would particularly warn you about such cases. Do not imagine the disease has become arrested and delay operation, but, on the other hand, urge most forcibly an immediate operation; for it may mean that a distended appendix has suddenly given way, and the sudden relief of tension has caused the temporary cessation of symptoms. Within a short time signs of spreading peritonitis will be present, or within a variable interval it will become evident that localised suppuration has occurred: which of the two will follow it cannot be foretold.

Asymmetry of the abdomen after an illness of two or three days' duration means abscess formation, although a swelling may not be detected on account of muscular rigidity. A palpable swelling when the illness has lasted three or four days also means abscess-formation.

It is by no means uncommon to find a very definite swelling, which is obviously an abscess, associated with diffuse peritonitis. I would warn you about such cases, for it is very easy to miss the diffuse peritonitis and merely concentrate the attention upon the localised abscess. There are many instances recorded in children where death has occurred more or less suddenly a few hours after an operation for appendicular suppuration. I am not referring to death from pulmonary embolism, which rarely occurs. The operation has passed off quite satisfactorily, and the child appears to have got over it. But it may be within twenty-four hours, or later, a more or less sudden collapse sets in, and in spite of all treatment death ensues within a few hours. My experience teaches me that some such cases are due to an unsuspected and entirely overlooked diffuse peritonitis, which was present at the time of operation. This more or less abrupt collapse followed shortly by death may occur during the course of any acute toxæmia. When following an operation it is thought naturally to be due to the operation, and so

it is in great part. The shock of the anæsthetic combined with that of the operation hastens the fatal ending in one already severely poisoned.

A rectal examination should never be omitted in any case of acute abdominal disease in the child. By this means it is possible to explore the whole pelvic cavity. I would remind you that the most frequent position of the appendix is one internal to the cæcum directed towards the pelvic brim and very commonly in part occupying the pelvis in the child. Even in the early stages of appendicitis, tenderness on the right side of the pelvis or an indefinite swelling suggesting matted intestinal coils in association with the symptoms are sufficient to warrant a diagnosis. In the later stages when abscess has formed in no less than six out of twenty-one cases this has been entirely within the pelvic cavity, and not palpable at all by the abdomen.

There can be no doubt that the only *treatment* of appendicitis is surgical. To my mind there is also no doubt that the operation should be performed as soon as the diagnosis has been made, at whatever period of the disease. I advocate, and practise whenever opportunity offers, immediate operation during the period of the minor manifestation of the disease. It is only by operating at this stage that the grave complications can be prevented. The only difficulty here is to persuade the parents to submit their child to an operation for such comparatively slight symptoms, symptoms from which the child has probably suffered previously and recovered quite perfectly. Before attempting to persuade the parents to the operation we must be convinced in our own minds of the diagnosis, and I feel certain that we may be by the signs I have mentioned. Although the symptoms may seem quite trivial, such is the beginning of a grave appendicitis in many cases, and we can never foretell the outlook in any case from the early signs, and we never know the condition of the appendix however slight may be the clinical symptoms. An operation conducted at this period is perfectly safe, and very much easier than one performed for localised suppuration, where it is sometimes by no means easy to find and remove the appendix.

Should diffuse peritonitis be present (and this refers not only to peritonitis of appendicular origin but all varieties), the first thing to do when preparations are being made for operation is to place the child in such a position that by gravity the peritoneal exudate will flow towards the pelvis. The pelvic peritoneum is more tolerant and absorbs less quickly than that of the upper abdomen. If transporta-

tion to a hospital or elsewhere be necessary this position should, if possible, be maintained. Secondly, an effort should be made to raise the blood-pressure. There is nothing so valuable for this as the infusion of saline solution. This should be administered subcutaneously. Thirdly, it is necessary to empty the rectum, as this cavity will be needed for the saline infusion after the operation. The operation must be conducted with all rapidity under an anæsthetic which produces the least degree of shock, and it may be necessary or advisable to administer saline infusion subcutaneously during the operation. The appendix must be removed and the peritoneal cavity drained. In the child it is nearly always possible to remove the appendix through a mid-line incision. The most essential drain, and practically the only one I use, is one to the bottom of the pelvis. Following the operation the position which allows gravitation towards the pelvis must be maintained, and rectal saline infusion at once commenced. The value of pituitary extract in combating the shock in these cases is, I think, very doubtful; I cannot say I have seen any good from its use in these cases in the child.

Before operation for localised suppuration, if the condition of the child demand it, saline solution should be administered subcutaneously. It is essential to empty the rectum as rectal saline infusion may be necessary afterwards. The not infrequent association of a local swelling and diffuse peritonitis must always be borne in mind, and if there is any doubt upon this point, after treating the local abscess it is much wiser to make a separate small mid-line incision and ascertain the condition of the peritoneum. Personally, I always aim at removing the appendix in all cases of abscess formation.

Pneumococcal Peritonitis.

To Bozzolo is given the credit of publishing the first case of pneumococcal peritonitis in 1885. This was a case of pleurisy and peritonitis, and from the exudate the pneumococcus was isolated. During the following few years several isolated cases were published by different observers. Nélaton, in 1890, performed the first operation for the disease without success in a woman, aged 32 years. Sevestre, in the same year, operated successfully upon a child for pneumococcal peritonitis. Cassaet, in 1896, was able to collect twenty cases from the literature. Michaut, in 1901, was able to collect thirty-three cases from the literature, and Jensen, in 1903, collected fifty-eight cases in children. Since that date the literature

upon the subject has increased enormously, showing that, as so frequently noticed in other cases, when a disease, however rare it was once considered, is described fully, the rarity becomes a fiction, and in reality the disease is comparatively common. This is so in pneumococcal peritonitis, for it is by no means a rare disease, and one that we may encounter any day. The pneumococcus is secondary to the appendix as a cause of peritonitis in the child.

There is no doubt that pneumococcal peritonitis is a much more frequent disease in early than in later life. Six out of seven of my cases have been in children. Probably the disease is twice or thrice as frequent in the child as in the adult. The difference in sex is peculiarly noticeable in children. All my cases have been girls. In fifty-eight cases in children collected by Jensen there was a proportion of seven boys to fifty-one girls. Lenormant found the ratio six to thirty-eight. Matthews says it is seven times more frequent in girls than in boys. In adults, on the other hand, the sexes seem to be more equally affected.

Pneumococcal peritonitis may be primary, *i.e.* the peritonitis is the sole or the predominating evidence of the pneumococcal infection, or it may be secondary to some distant focus of infection. In one of my cases the peritoneum, both pleuræ and the pericardium were infected, and although the disease was far more extensive in the peritoneum, it cannot be definitely asserted that this was the primary lesion. In my other cases the peritonitis was the sole lesion, and in children, in the great majority of cases, we have to do with a primary pneumococcal peritonitis. Occasionally, however, the peritonitis is associated with pneumonia or pleurisy, and in some of such cases the peritonitis appears to be the primary lesion, whereas in others it may be secondary to the pulmonary infection. When secondary to a pulmonary affection it has been explained by a direct trans-diaphragmatic spread. Such, however, is probably not usual, for the absorption from the pleura is greatest in the mediastinal and costal pleuræ, and is comparatively little in the diaphragmatic pleura, and the lymph-flow through the diaphragm is from the peritoneal to the pleural aspect; a lymphatic spread of the pneumococcus from the pleura to the peritoneum implies a flow of the lymph-current contrary to normal. It has, however, been proved, both experimentally and from the examination of persons who have succumbed to pneumonia, that the spread of infection from the pleura to the peritoneum does occur. Pneumonia and pleurisy, both extremely common in children, are rarely complicated with peritonitis.

Since a pulmonary infection so infrequently precedes the peritoneal, and when it does, seeing how improbable it is that the infection of the peritoneum is directly infected by the trans-diaphragmatic route, we must consider other means by which the pneumococcus may reach the peritoneum. Three ways suggest themselves: by the blood, from the gastro-intestinal tract, and *viâ* the genital organs.

By the blood.—The pneumococcus is constantly found in the mouth; it frequently becomes pathogenic, producing middle-ear disease, angina, etc., and it is conceivable that it may enter the blood and be carried to the peritoneum. This may be the path of infection in the cases secondary to pneumonia or pleurisy, for it is stated that the pneumococcus is frequently found in the blood of patients the subject of pneumonia. An infection by the blood to the peritoneum has, I believe, never been demonstrated; it is purely hypothetical.

By the gastro-intestinal tract.—This seems certainly the most likely route. The pneumococcus is constantly present in the mouth, and, therefore, is presumably frequently swallowed, and enters the stomach. It is stated that it is killed by the acid of the stomach, but probably it may escape and become pathogenic in the bowel. The pneumococcus has been found in association with various pathological lesions in the intestines. It has been found in some cases of enteritis, in some cases of gastritis, in some cases of perforated gastric ulcer; it has been found in abscesses in the intestinal wall, and by no means rarely in disease of the appendix. Clinically, this route of infection is supported, since gastro-intestinal symptoms are constantly present. Pathologically, on the other hand, it is extremely rare to find any coarse lesion of the gastro-intestinal tract in children who succumb to pneumococcal peritonitis.

By the genital organs.—This route is suggested by the strikingly greater frequency of the disease in girls than in boys. In adults the pneumococcus has been found, in association with other organisms, in cases of pyosalpinix, endometritis and vulvitis, but in children I believe it has not yet been possible to trace the infection to the peritoneum by this route. Nevertheless, a detailed microscopical examination might reveal some evidence in the future, for there must be something in this great disproportion of sex-incidence.

Clinically pneumococcal peritonitis in its ultra-acute form differs in no way from any form of diffuse septic peritonitis. Operation is undertaken upon the belief that it is the latter variety of peritonitis which is present, and only the bacteriological examination of the

exudate will settle the diagnosis. When operating upon a case of diffuse peritonitis for which no obvious cause can be found, the pneumococcus should always be thought of, for it is possible that a serum or a vaccine may be of use should it prove to be pneumococcal in origin. This acute form of the disease is by no means an infrequent variety in the child.

When the disease is less acute the exudate may be encysted or diffused. The initial symptoms are similar in each case. The onset is quite abrupt with abdominal pain, vomiting and fever. The pain may be localised or diffused, and, if localised, is generally referred to the lower abdomen. The fever is variable, but is always present. Rigidity of the abdominal muscles in some degree is always present, but it may be less evident than in the insidious type of appendicular peritonitis. Within a few hours, or perhaps not for a day or two, diarrhœa sets in, resisting all treatment. The ejecta are always foul-smelling. This diarrhœa is so constant as to form one of the features of the disease, and, indeed, it seems to be the most characteristic. Diarrhœa may occur in any form of peritonitis, especially that of appendicular disease in children, but this differs from the foul diarrhœa of pneumococcal peritonitis. After twenty-four or forty-eight hours some amelioration of symptoms occurs. The vomiting ceases, the temperature falls, and the general condition is decidedly improved. The temperature, however, never reaches the normal line, but oscillates a variable distance above this. Abdominal pain never disappears, neither does rigidity of the abdominal muscles. If the inflammation is diffusing the pain, tenderness and muscular rigidity will become more wide-spread; the abdomen will distend, and the aspect of the child will alter. If allowed to progress, signs of free fluid in the peritoneal cavity may be detected. Showing how subacute the course of this disease is in some cases, I may mention that children have been operated upon successfully on the eighth, tenth, and thirteenth days of the illness. If the exudate becomes encysted, after a variable interval following upon the initial symptoms a swelling is detected in the abdomen. This swelling is almost invariably in the lower abdomen, frequently rising out of the pelvis; sometimes more towards the right side, and occupying the right iliac fossa. It may not be for some days that this swelling makes itself evident: indeed, it has generally been recorded as occurring about the tenth or twelfth day of the illness.

Such is the clinical course of pneumococcal peritonitis. There is nothing distinctive upon which a diagnosis can be made, unless it be

the foul diarrhœa, and this is probably merely suspicious. It is frequently mistaken for appendicitis, and the more subacute forms for typhoid fever or tuberculous peritonitis. There should, however, be no difficulty in recognising the case as one of peritonitis, for which condition surgical treatment is necessary. If an early operation be undertaken in all probability there will be nothing distinctive in the exudate, appendicitis will be diagnosed and this organ removed. Should this be found healthy, pneumococcal peritonitis should at once be suspected. In the subacute cases the character of the exudate and the lymph will generally serve to distinguish pneumococcal peritonitis. The treatment after operation is similar to that of any variety of peritonitis. The rectal infusion of saline solution is essential to combat shock, and in addition it tends to render more fluid the thick, sticky peritoneal exudate, and thereby allows its more ready escape through the drainage-tubes.

(*To be continued.*)

TUBERCULOSIS OF THE BRAIN WITH INVOLVEMENT OF BOTH OPTIC THALAMI.

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A girl, aged 10½ years, was admitted to the Royal Hospital for Sick Children, Glasgow, on March the 15th, 1911, with the following history: Eight months prior to admission she had an acute febrile attack, which was accompanied by epistaxis and headache, partly occipital and partly vertical. The acute symptoms subsided, but she never became quite well; the headache persisted, and she began to lose flesh. Three months later, subsequent to the removal of tonsils and adenoids, she had a very severe inflammation of the throat with high temperature. Shortly afterwards there was a discharge of pus from the right ear. In the course of a week or two the fever subsided and the throat got better, but the ear continued to discharge for a time, and her general condition got gradually worse. She remained in this state until a fortnight before admis-

sion, when she had a return of acute symptoms characterised by fever and an exacerbation of the headache, which was mainly occipital in type. She now lost flesh rapidly and sweated a good deal at night. A constant fine tremor then gradually made its appearance in the limbs, especially in the fingers. The patient became much weaker, but did not show signs of mental dulness. She never had any vomiting, head-retraction, convulsions, or squint.

There was nothing of importance to note in the child's previous health. The family history was good, there being no suggestion of tubercular or specific disease.

Condition on admission.—Temperature 100° F., pulse 120 and respirations 24 per minute. The patient was very emaciated. Her face was flushed, but there was no lividity. The general intelligence did not appear to be impaired, but the child was very irritable and screamed whenever she was touched. A special feature of her condition was the presence of a generalised tremor producing a state closely simulating a rigor, but the temperature was not greatly elevated and did not subsequently rise. This tremor was constantly present unless when the child was asleep, but was most marked when she was being examined or being attended to by the nurse. In the limbs the tremor was fine and rhythmical, but was more distinct in the arms and hands than in the legs and feet. In the neck and trunk the tremor was coarser in character. The lower jaw was also in constant movement. There was no loss of volitional or emotional mobility of the face. Retraction of the head was absent, but the posterior cervical muscles were contracted and painful. There was no rigidity or paralysis of the limbs, but Kernig's sign was found present to a slight extent. The knee-jerks were active, but not unduly so; ankle-clonus could be elicited in both limbs; the plantar reflexes were flexor in type. No disturbance of sensation could be detected, but this part of the examination was not satisfactory.

The eyes.—Both pupils were moderately dilated and reacted only sluggishly to light. There was no strabismus nor nystagmus. Double optic neuritis was present. Lumbar puncture was performed, but the fluid obtained was quite clear, and the deposit obtained after centrifugalising was found microscopically to consist of normal cells. No micro-organisms could be found. The ears were examined, but showed no active disease, though there was evidence of past mischief in both, especially the right.

Thoracic and abdominal organs.—Respiration was a little rapid, otherwise it was undisturbed. The lungs revealed no abnormality to

percussion or auscultation. The examination of the circulatory system revealed nothing abnormal beyond a slight undue rapidity of the pulse and weakness of the heart's action. The tongue was moist and thickly coated with a white fur. A generalised tenderness was complained of in the abdomen, but nothing abnormal was palpable. The splenic dulness was slightly increased in size, but the organ was not palpable.

Progress.—After admission the child remained in practically the same condition for a week. The temperature showed daily variations ranging from about 100° F. to normal. The pulse varied in rate considerably, but was never very slow and never showed irregularity. The tremor persisted in the form already described. Seven days after admission there was a distinct change for the worse. She seemed less intelligent and frequently cried out as if in pain, particularly on any attempt being made to move her, but no new localising symptoms developed.

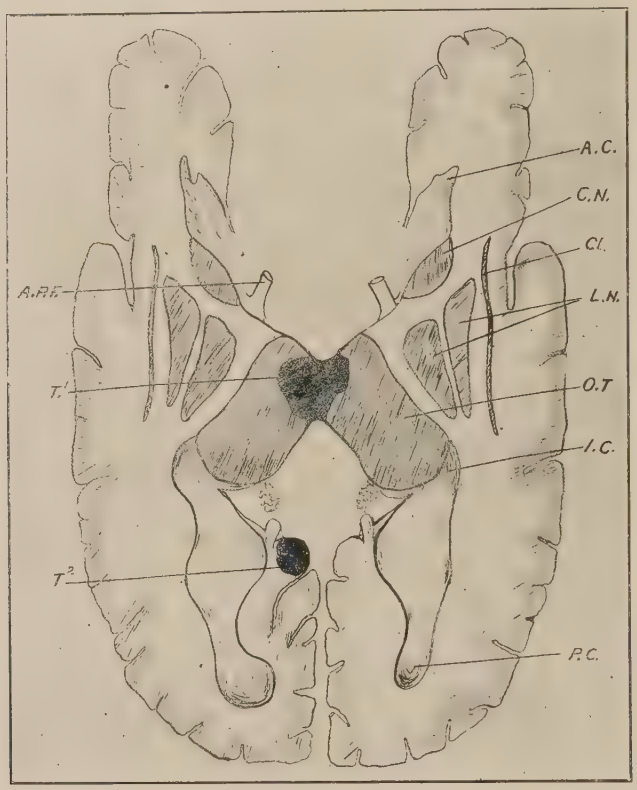
On the evening of March the 25th, ten days after admission, there was a sharp rise of temperature to 102·4° F. followed by a sudden fall to normal. A further rise occurred the following morning to 101·2° F. The child became gradually semi-comatose and passed urine and fæces in bed. The tremor still persisted but was less marked. There was now distinct impairment of the percussion resonance over the upper part of left lung, and in this situation the respiratory murmur was diminished in volume. The condition of the patient now got rapidly worse, and she sank into an unconscious state. The breathing became short and laboured and the pulse got more rapid and feeble. The tremor practically disappeared, as did also Kernig's sign. Definite râles made their appearance over the upper part of the left lung. Just before death there was a more flaccid condition of the left arm than of the right.

On March the 26th lumbar puncture was again performed; the fluid would not flow, though about one drachm of clear fluid was withdrawn by a syringe. The child died on the following day.

Post-mortem examination.—On exposing the brain after the dura mater had been removed, well-marked tubercles were found in the sulci over the vertex, and the convolutions were seen to be flattened. On removing the brain a small nodule was left adherent to the dura on the left side just below the lateral sinus. This was found to be a small tumour, about the size of a small hazel-nut, which had shelled out of a cavity in the left lobe of the cerebellum. There was a well-marked tuberculous meningitis involving the base of the brain. On section of the brain the lateral ventricles were found to be enormously

distended with clear fluid. On reaching the basal ganglia a tumour was found occupying the site of the massa intermedia between the two optic thalami and partially involving both these ganglia.

On making further sections this tumour was found to occupy the



Horizontal section of brain through the basal ganglia, showing position of tumour. The anterior portions of the cerebral hemispheres are represented as lying too widely apart, due to a separation that occurred during hardening of the specimen. A. C. Anterior cornu of lateral ventricle. I. C. Inferior cornu of lateral ventricle. P. C. Posterior cornu of lateral ventricle. C. N. Caudate nucleus. CL. Claustrum. L. N. Lenticular nucleus. O. T. Optic thalamus. A. P. F. Anterior pillar of fornix. T^1. Tumour in optic thalami. T^2. Tumour in gyrus lingualis of left occipital lobe.

greater part of the anterior portion of the optic thalamus on the left side, and also, though to a less extent, the same part on the right side (see diagram). Another tumour was found in the gyrus lingualis of the left occipital lobe, and still another was found in the left lobe

of the cerebellum just anterior to the site occupied by the one already referred to.

A generalised miliary tuberculosis was found in both lungs, with some broncho-pneumonic consolidation in the upper lobe of the left. Bronchial glands were enlarged, and some of them were caseous. Miliary tubercles were present also in the liver and spleen. The kidneys were quite healthy. No tubercle was seen in the peritoneum or bowel, and the mesenteric glands appeared to be free. Microscopical examination of one of the tumours showed it to be typically tuberculous with caseation in the centre surrounded by active tuberculous tissue containing giant-cells.

Remarks.—The above case is interesting on account of the generalised tremor, so marked a symptom during life, being associated with a well-defined lesion of the optic thalami as proved by post-mortem examination. Whether it would be correct to ascribe the tremor to this lesion is worthy of some consideration. The lesion of the optic thalami was not the only cerebral lesion. There were other small tumours present, but these were so situated as to be easily excluded as a cause of this symptom. Tuberculous meningitis was also present, but the tremor was probably not due to this cause. Generalised tremor present continuously for many days is not in our experience a feature of meningitis. Muscular twitchings in this condition are common, and a rapid rhythmical tremor, especially of the arms, is said to be an occasional symptom, but in both cases they are either fleeting or of comparatively short duration.

It seems, therefore, reasonable to consider the involvement of both optic thalami as being the lesion accounting for the generalised tremor.

The functions of the optic thalami are not yet fully understood. They are generally believed to receive the stimuli which flow by the sensory tracts from the periphery and to transmit them to the cortex of the brain. Yet hemi-anæsthesia has not been proved to be a definite result of a lesion of the optic thalamus, just as a lesion of the pulvinar, the part of the thalamus which receives numerous fibres of the optic tract, does not inevitably give rise to hemianopsia, though a lesion of the external geniculate body will do so.*

Another function of the optic thalami is said by many to be that of acting "as a centre for involuntary automatic movements for the

* 'Text-book of Nervous Diseases,' by Prof. H. Oppenheim, of Berlin. Fifth edition; authorised translation by Alexander Bruce, M.D., F.R.C.P.E., LL.D.; Edinburgh, 1911; p. 650.

psycho-reflexes and therefore for movements not directly controlled by the will. Lesions of the thalamus, therefore, will, according as they have an irritating or a paralysing effect, produce either exaggeration or abolition of the automatic mimic movements."* It has already been noted that no disturbance of this kind was detected in our case.

Again, involuntary movements, such as hemichorea, hemiathetosis, and tremor, especially as post-hemiplegic phenomena, have been observed when the optic thalami were involved. In our case we have tremor associated with involvement of the optic thalami but independent of any hemiplegia. The interpretation of such involuntary movements has given rise to many theories.

"Are we, in fact, dealing with an irritation of a centre for involuntary movements? Or does the irritation of sensory fibres to the cortex cause stimulation of the motor regions which manifests itself in these involuntary movements? Are these fibres from the thalamus to the cortex for the inhibition of the functions of the motor region, whose interruption causes these symptoms of motor excitement, etc.? Or does the impulse from them arise, not from the optic thalamus, but rather from simultaneous lesions of an adjacent tract?"*

These remarks of Oppenheim show that the explanation of the tremor, even if we accept it as being connected with the lesion in the optic thalami, is a matter of speculation. The simplest view would be that the involvement of both optic thalami in a degenerative lesion produced an irritative effect upon the internal capsules; and the portions of these chiefly affected would be the genu and the anterior part of the posterior limb—parts occupied by the motor fibres passing to the mouth and upper limbs. Less affected would be the part further back containing the fibres going to the lower limbs. This would explain the distribution of the tremor, the arms being more affected than the legs. The sensory fibres, which lie most posteriorly as we have seen, were probably not affected.

This case is peculiar in the fact of both optic thalami being affected; hence the plausibility of explaining the bilateral tremor by this lesion. In this case, as in many other cases where the optic thalami have been involved, the symptoms produced are probably the result of the implication of adjoining structures. We are thus drawn to the conclusion that the bilateral tremor in this case was due to irritation of the motor tracts in the internal capsules by the presence of the tumour in the optic thalami.

* *Loc. cit.*, p. 651.

Royal Society of Medicine.

SECTION FOR THE STUDY OF DISEASE IN CHILDREN.

Provincial Meeting, held at Addenbrooke's Hospital, Cambridge, on July the 8th, 1911.

Dr. E. CAUTLEY, *President, in the Chair.*

Cretinism.—Dr. E. J. CROSS.—Boy aged 6 years. First seen in March, 1910; then said to be suffering from kidney disease. The eyelids were then very puffy and swollen, the skin dry and scaly, and the hair thin. There was great muscular weakness and mental deficiency. Height 41·5 in. In June, 1911, the boy was much brighter and took an interest in life. Height 48 in. Treatment: Thyroid extract, 1½ gr. at bedtime, increasing to 3 gr. daily.

Cretinism.—Dr. J. ALDREN WRIGHT.—Boy, aged 15 years. At birth apparently normal. Did not walk until 3½ years old. When aged 7 years mother noticed that he was stunted in growth. Condition when first seen, March, 1910, aged 14 years: Height, 30 in.; weight, 31 lb.; intelligence appeared absent; rarely moved; could neither walk nor talk. Skin dry and wrinkled; hair scanty. Large supra-clavicular and anterior axillary pads. Thyroid not discernible. Temperature, 97·6° F. Was given thyroid extract. Since then there had been a marked and continued improvement in intelligence and appearance. Present height, 39 in.; weight, 35 lb.

Congenital Heart Disease; Cerebral Hemiplegia.—Dr. J. ALDREN WRIGHT.—Boy, aged 7 years. Nothing wrong noticed until 18 months old, when, having previously walked well, "his left leg began to give way," causing him to fall at times. At this time fits also began to occur. The child first came under observation when he was 2 years and 11 months old. He was having twelve fits daily, and could neither sit up nor stand. Intelligence considerably impaired. Very passionate. Both knee-jerks were exaggerated. Plantar reflexes extensor. No ankle clonus. Left leg smaller than right. Systolic murmurs heard at apex, but loudest at pulmonary area (pulmonary stenosis). Present condition: Fairly well-developed boy; left side smaller than right. Intelligence and temperament improved. Knee-jerks exaggerated. Plantar reflexes flexor. Fits now occurred only during sleep—day or night; and, recently, only once daily.

Cerebellar ataxia.—Dr. J. ALDREN WRIGHT.—Girl, aged 14 years. When 4½ years old she somewhat suddenly lost the use of her legs, then of arms and speech. She had difficulty in swallowing. No control of her sphincters. No knee-jerks. Plantar reflexes flexor. After a time improvement set in; first beginning to crawl, then to walk, recovering control of her sphincters, etc. Present condition: Mental condition still impaired; in infant class at school. Talks slowly, but fairly distinctly. Sallow and expressionless countenance. No fits. Temperament improved; screamed

less than formerly. No nystagmus. More sensitive to touch and alterations of temperature. Walked with reeling gait and feet apart. Most unsteady in starting to walk. Knee-jerks absent. Plantar reflexes flexor. Abdominal reflexes active. Some inco-ordination of hands.

Congenital Nævoid Condition of the Left Thigh.—Mr. W. H. Bowen.—Boy, aged 12 years, perfectly healthy and leading an ordinary life. Over the left buttock and outer side of thigh, irregularly arranged, was a surface portwine-like stain, but rather lighter than that usually seen. On the thigh the condition was associated with plexiform subcutaneous veins. The left leg was considerably larger in girth than the right, the measurements round the limb at the thickness of the calf being 13 in. and 12 in. respectively, and just above the ankle $7\frac{7}{8}$ in. and $7\frac{5}{8}$ in. Save for a small patcho f cutaneous staining just below the knee there were no visible external signs of the leg or foot being affected in the same way as the thigh.

Glandular Fever.—Mr. W. H. Bowen.—A temperature chart showing fluctuations in temperature in a case of enlargement of lymphatic glands in neck following measles, and shown as the most pronounced example of this type of "glandular fever," of which several cases were seen. The main points of the cases were that they followed attacks of measles at varying intervals, they started as a cervical adenitis and peri-adenitis with variable fever, that the constitutional conditions were never severe, and that complete recovery occurred without any suppuration in any case. It seems probable that the measles prepared the soil, and that a slight tonsillar inflammation led to the secondary glandular change.

Hydrocephalus and Buphthalmos.—Dr. J. C. W. Graham.—Boy, aged 3 years. Right eye buphthalmic at birth and removed. Left eye gradually became buphthalmic, and was removed in March, 1910. The head measured 20 in. in circumference in December, 1909, and now measured 22 in.

Congenital Cataract.—Dr. J. C. W. Graham.—Girl, aged 9 years. Congenital central deposit posterior surface of each lens. Vision $\frac{6}{12}$ in each eye.

Hypermetropic Astigmatism with Macular Changes.—Dr. J. C. W. Graham.—Girl, aged 8 years. Vision in each eye $\frac{6}{36}$ in April, 1910; reduced to board only in March, 1911. Slightly improved by glasses.

(*To be continued.*)

CLINICAL SECTION.

Friday, May the 12th, 1911.

Foreign Bodies Removed from Œsophagus.—Mr. Herbert Tilley showed (*a*) a teat of a "comforter" removed from a child, aged 4 days, by direct œsophagoscopy; (*b*) a penny which had been impacted thirteen

days removed from a girl, aged 13 years; (*c*) a farthing which had been impacted for five hours removed from a boy, aged 5 years; (*d*) a safety-pin removed from a child, aged 3 months.

Graves's Disease in a Boy.—Dr. W. Essex Wynter showed a boy, aged 11 years, whose eyes had become unduly prominent nearly one year previously. In addition to proptosis there were general enlargement of the thyroid, fine tremor of the fingers, and a pulse of 92 to 124. The skin was moist and flushed, and there was yellowish pigmentation of the hands and neck. There was incontinence of urine and slight albuminuria. Thyroidectin had been given in 5-gr. doses thrice daily.

Ectopia Vesicæ in a Female Child.—Mr. E. Musgrave Woodman showed a rickety child, aged 2 years, with congenital ectopia vesicæ. The symphysis pubis was wanting and the pubic bones badly formed. The vagina and rectum were normal. In 1906 Trendelenberg could find only thirty-five cases of ectopia vesicæ in females on record. Mr. Woodman was in favour of extra-peritoneal engrafting of the bladder into the rectum.

Congenital Deformity of Femur.—Mr. Frank Kidd exhibited a child, aged 1 year and 5 months, in whom the X rays showed an absence of the upper epiphysis and of half the shaft of the left femur. There were no other deformities.

Philadelphia Pediatric Society.

Meeting, May the 9th, 1911, J. Torrance Rugh, M.D., President.

A CONSIDERATION OF THE CAUSES OF THE HIGH INFANT MORTALITY DURING THE SUMMER MONTHS.

Transmission of Infections.—Dr. Allen J. Smith, by invitation, read this paper. After indicating that in any case the transmitting agency of an infection was to be referred to either a vital bearer or to some non-vital substance upon, or in which, the infection might happen to be present, and after stating that in a broad way the infant mainly owed its well-known relative freedom to infection in the first months of life to its isolation, Dr. Smith took up in turn a number of the more striking illustrations of living and non-vital bearers of infection. Of all animal conveyers man was himself the most common transmitter of infections to the infant. Therefore Dr. Smith urged the restriction, as far as possible, of indiscriminate fondling of infants by others than proper caretakers. Animal household pets, particularly cats and dogs, were held responsible not infrequently for the infection of children as well as adults, especially in connection with ringworm and analogous skin infections. Insect transmission of infections occurred in infancy and childhood precisely as in later life, the accounts of Koch, Wellman and others in reference to the prevalence of malaria in early life in Africa and of Guitéras in reference to yellow fever among children being illustrations to the point in case

of mosquitoes. Fly-borne ophthalmias, suppurations and diarrhœal affections (with reflection on the probability of a higher rate of typhoid fever prevalence in the first few years of life than was generally believed, and the likelihood of its transmission by house-flies) were instanced as examples of conveyance by this class; and the same possibility of other arthropods transmitting disease to infants and children as to adults was insisted upon under appropriate conditions of access. Dr. Smith referred to Patten's work upon bed-bugs as intermediate hosts of Leishman-Donovan bodies and to personal communications from Campbell, of San Antonio, Texas, upon bed-bugs as possible purveyors of smallpox. Leaving, of the inanimate conveyers, the general consideration of milk and other foods for the subsequent speakers of the evening, and indicating the unlimited possibilities of acquirement of various infections from all sorts of objects with which the child was constantly coming in contact, Dr. Smith laid especial emphasis upon the possibility of easy acquirement of infectious organisms and parasites or their ova from floor contact when the infant reached the crawling stage of its life. A striking clinical illustration of contact infection was mentioned in the case of a child about one year of age, observed by Professor M. B. Hartzell in the Dermatological Dispensary of the University Hospital, and by him mentioned to Dr. Smith. The child had well-developed lupus lesions on the hypothenar eminence of one hand and on one knee. Suspecting from the situation of the lesions that it had been acquired from tubercle bacilli rubbed into the parts from the floor on which the child crawled, Dr. Hartzell readily elicited the fact that the child usually spent a large part of its waking hours on the floor of the room in which its father was confined to bed with advanced pulmonary tuberculosis, and that the child's mode of crawling was definitely on hands and knees. The adherence of bacteria, encysted protozoa, ova of intestinal parasites, as of *Hymenolepis nana* from mouse-droppings, along with harmless dust particles, to the moist and sometimes sticky hands of the crawling infant from the dirty floor, their ready transmission to the alimentary canal when the hands went to the mouth, the infection of the skin of the face and scalp when the dirt was rubbed into these parts, were referred to; and brief reference to acquirement of disease from the ground outside of the house was made. Soiled and infected clothing, bedding and toys as articles liable to convey disease were considered. In closing, Dr. Smith, while acknowledging the possibility of carrying his argument to a ridiculous grade of impracticability, insisted that the keynote of avoidance of infectious and parasitic disease rested in the grade of isolation of the infant and the cleanliness maintained in its surroundings.

Dr. Samuel McC. Hamill read a paper on **How Physicians can aid in Establishing Conditions to Control the Milk Supply, as Recommended by the Mayors' Milk Commission.**

The Relation of Milk to Infant Mortality.—Dr. H. W. Conn, of Wesleyan University, by invitation, read this paper. He said that all dangers in milk were connected with its bacterial content. Milk was not necessarily harmful because it contained very large numbers of bacteria, for buttermilk was known to be good and it had millions of germs in it. But the presence in milk of certain bacteria, those of tuberculosis, typhoid fever, diphtheria and scarlet fever, constituted a great danger.

The occurrence of any of these infections in milk was always due to careless handling of the milk. Besides these, indefinite intestinal troubles of infants in hot weather, characterised chiefly by diarrhœa, were due to bacteria in the milk, for they were more common among children fed on cow's milk, they were controlled by sterilising the milk, they were most common in hot weather, when bacteria in the milk were most numerous, and they might be greatly reduced by rigid milk inspection. Certified milk was beyond the means of the masses. Pasteurisation, properly performed, came nearest to the solution of the milk problem. The milk was thereby kept at 140° F., not higher, for half an hour. It did not change the digestibility or the taste of the milk, but destroyed all pathogenic bacteria.

In opening the discussion Dr. C. J. Hatfield said that the paper by Dr. Conn had been to him a liberal education upon the milk question. He did not feel qualified to discuss it beyond expressing his interest in, and admiration for, the work which Dr. Conn had carried out. As to the coming milk show, which he had been requested to speak of, he believed that it was the direct result of the work done by the Milk Commission of the Philadelphia Pediatric Society. He traced the influence of this work to the appointment of the Mayor's Milk Commission, the report of which, made public in February, marked a great advance in the campaign for better milk for the city. The milk show was a direct result of this work. The plan was initiated by the Director of Public Health and Charities and the Chairman of the Milk Commission of the Pediatric Society. An efficient organisation had been formed, and a number of committees were working splendidly with every prospect of a successful exhibition. The milk show would be held at 809, 811, and 813, Chestnut Street, from May the 20th to the 27th. There would be an exhibit of dairy productions with models; the care necessary to proper handling of the milk would be shown by photographs; the care of the milk in the home, in cooking, etc., would be demonstrated; moving pictures would be shown at intervals, indicating the transformation of an old-fashioned, dirty dairy to a modern up-to-date plany; exhibits from the United States Department of Agriculture, from the Laboratory of the Department of Public Health of the City and from the Veterinary Department of the University of Pennsylvania would be shown. There would be commercial exhibits of appliances used in caring for milk, also samples of different grades of milk. The programme of lectures and demonstrations, given at noon, at three, and at eight o'clock each day, was most impressive. The lecturers included the most prominent men interested in the production of milk from all parts of the United States. Additional teaching in the subject would be given in the Dairy Institute, to be held at the University of Pennsylvania during three days of the Show week.

Dr. A. C. Abbott said that he objected to depending upon bacterial counts as tests for milk, since results were obtained after the milk had been consumed. He preferred microscopic examination of the milks after centrifugalising, so that fifty or more examinations might be made in a morning. By this means many indefinite infections could be discovered. Dr. Abbott had opposed pasteurisation as he considered it an obstacle to obtaining clean milk from healthy cattle. Five years ago no pasteurising plant in this city was turning out milk that was not re-infected after having been pasteurised; that was probably true still. The damage done to milk was always due to ignorance, both of the producer and of the consumer. Dr. Abbott thought that Philadelphia had as good a milk supply as any of the large cities. It is only a question of time until the recommendations made by the Mayor's Milk Commission would be met.

In closing the discussion Dr. Conn stated that he did not believe that milk inspection would eliminate tuberculosis from milk until the time when all cattle could be tested for tuberculosis, and that was a long distance ahead. In the meantime they had to face the problem of what to do before that time came. He did not believe that milk inspection or dairy inspection would ever be able absolutely to eliminate the possibility of the distribution of typhoid fever or other contagious diseases distributed by milk. It was a consideration of these facts that led him to accept pasteurisation.

Société de Pédiatrie, Paris.

May the 16th, 1911. (*Bulletin No. 5.*)

Bronchio-tracheal Adenopathy.—M. H. Barbier, continuing the discussion on hypertrophy of the thymus, related the case of a boy, aged 3 months, who had attacks of suffocative cough, recession of thorax, dysphagia, dulness over sternum, and a radiographic shadow in that locality. Tuberculous glands and mediastinitis were found at the autopsy The author was of opinion that operation is always contra-indicated on account of the presence of mediastinitis in such cases.

Microscopic Examination of the Cerebro-spinal Fluid in Tuberculous Meningitis.—MM. A. Netter and A. Gendron stated that Nageotte's method (see last 'Bulletin') gave better results than the examination of the clot obtained by centrifugalisation. In tuberculous meningitis the proportion of cellular elements varied from 60 to 300 per c.mm.; it was usually less in poliomyelitis. The injection of serum did not modify the leucocytic formula in tuberculous meningitis, while a marked change was observed in cerebro-spinal meningitis or poliomyelitis. Koch's bacillus could be found twelve times out of fourteen examinations after the first puncture, but for this the clot of centrifugalisation and the fibrinous adherent deposit must be examined. Cerebro-spinal fluid formed an excellent culture medium for the tubercle bacillus

The Respiratory Index in Nasal Insufficiency in Childhood.—M. Prosper Merklen showed how the measure of the axillary perimeter in respiration and inspiration was capable of giving valuable information in children who were subject to cough. Diminution of this respiratory index is in favour of nasal insufficiency, although the condition of the thoracic and abdominal viscera was normal and there was no tracheo-bronchial adenopathy.

Fulminating Meningeal Hæmorrhage in a Boy, aged 17 years, the Subject of Glandular Tuberculosis.—M. L. Guinon related this case, which had been under his care since the age of four years. The only symptoms were loss of weight, nervousness, malaise and headache. The physical signs were harsh prolonged expiration at the apices and an extra-cardiac murmur at the base of the heart. Returning home after this last

examination the boy became semi-comatose. Two hours later the coma was profound, the pulse rapid and rhythmic. Lumbar puncture drew off a bloody fluid under tension; death took place shortly afterwards. The author was of opinion that there was a tuberculous lesion of an artery which ruptured during the respiratory efforts made during examination.

VINCENT DICKINSON.

Institut de Puériculture, Paris.

AT the inaugural ceremony on June the 8th Dr. Variot delivered an eloquent address before a distinguished audience. The Institute is situated within the grounds of the Hospice des Enfants Assistés, in which Parrot carried out his well-known work on athrepsy, and was succeeded by Prof. Hutinel, Dr. Variot's immediate predecessor. The Institute is to serve two purposes: first, as a centre for scientific study and research, and secondly, as a school for the popularisation of infantile hygiene. The first object is to be attained by utilisation of the unrivalled clinical material within the hospital, which is also provided with special laboratories for pathological anatomy, bacteriology and radiology. Another laboratory for milk investigation is soon to be added. The second object Dr. Variot hopes to realise by the establishment of a milk *dépôt* similar to the "Goutte de Lait," which he has directed for many years at Belleville, and by the institution of a regular course of lectures, including practical demonstrations to mothers, school teachers, and girls above the age of twelve years.

Abstracts from Current Literature.

Medicine.

Diphtheria in an infant (*'Arch. f. Kinderheilk.,'* 1910, LIV, *p.* 182).—**G. Wolkenstein** was called to see a child aged 7 days, who for the last forty-eight hours had begun to cry as soon as it was put to the breast. Membrane was found on both tonsils. There was slight respiratory obstruction. Rectal temperature 100°. Fifteen hundred units of antitoxin were injected, and camphor and valerian were given internally. Diphtheria bacilli were subsequently found. On the day following the injection the membrane had almost disappeared, the pulse and temperature were normal, and the child took the breast well. Recovery occurred without complications.

J. D. ROLLESTON.

Diphtheria of vulva (*'New York Med. Journ.,'* 1911, I, p. 24).—**L. L. Smith** was called to see a girl, aged 3 years, who could not pass urine. The orifice of the urethra and the labia minora were inflamed and swollen. Urine removed by catheter was normal. Temperature 100° F. Fauces and nostrils clean. The next day there was slight muco-purulent

vaginal discharge, examination of which showed an absence of gonococci. The following day diphtheria bacilli were found in pure culture in a smear taken from the labia minora: 2000 units were injected, and within twenty-four hours the urinary symptoms had begun to subside; 5000 units were subsequently given, and in a day or two the symptoms had entirely subsided. A greyish-white membrane separated from the nymphæ and labia majora. Subsequent recovery was uneventful. The child had probably been affected by using the same bath-towel as a visitor who had been exposed to diphtheria, and had been suffering from a "cold" during her stay.

J. D. Rolleston.

Primary diphtheria of the external urinary meatus (*'Practitioner,'* 1910, II, *p.* 724).—**Alfred Howell** records a case of the above in a little girl, aged 9 years. The child was brought for advice regarding what the mother thought was a "blood-clot" in the front passage. Local examination revealed a purplish, angry-looking swelling, occupying the position of the external urinary meatus and its immediate neighbourhood, rounded in outline, reaching below the base of the hymen and completely hiding the latter. The centre of the swelling was depressed and admitted with difficulty a small catheter. Urine was drawn off and showed nothing abnormal. Several punched-out ulcers were to be seen around the margin of the depression, and a thin, sanious discharge exuded from the whole surface; no false membrane was seen. Constitutional symptoms were absent, and while the swelling was tender to the touch there was no complaint of pain, even during or after micturition. There was no history of any local injury or previous disease. Rest in bed, a sedative lotion locally, and an alkaline mixture internally failed to effect any improvement. Bacteriological examination demonstrated the presence of the Klebs-Loeffler bacillus. Two-thousand units of antitoxin were injected and the condition subsequently quickly cleared up. Apart from slight redness of the part, which persisted for some time, the child showed no complications or sequelæ. The author thinks the following points worthy of note: (*a*) the source and localisation of the infection; (*b*) the absence of constitutional symptoms; (*c*) absence of pain and presence of relatively little tenderness.

J. Allan.

Unusual localisations of diphtheria (*'Le Méd. Prat.,' July* 26, 1910; *abst.* *'Med. Rev. of Rev.,'* 1910, *p.* 606).—**Terrien** has called attention to an unusual localisation of diphtheria. He refers to the involvement of adenoid vegetations in the pharynx in the diphtheritic process. The following symptoms were noted: There was a marked coryza with a profuse discharge, which soon became sanious; there followed symptoms of croup and of diphtheritic bronchitis with progressive and continuous dyspnœa. In addition there was interference with nasal respiration, hoarseness, pain in the ear, swelling of the glands in the neck, a rapid pulse, and great prostration. In the presence of such symptoms diphtheria should be suspected, cultures taken from the pharynx and the nose, and, if possible, rhinoscopy performed.

J. Allan.

Relapse of diphtheria after measles (*'Bull. et mém. de la Soc. Méd. des Hôp. de Paris,'* 1911, XXXI, *p.* 254).—**L. Martin** and **H. Darré**.—A boy, aged 3 years, was admitted to hospital on January 11, 1911, with mild laryngeal diphtheria, which was treated with antitoxin, and was discharged on the 28th. On February 12 he developed measles, and on the 15th severe faucial

diphtheria, for which he was again injected. General urticaria appeared on the 16th, and a mixed urticarial and scarlatiniform eruption on March 2. The relapse of diphtheria is attributed to the fresh lesions of the faucial and laryngeal mucosæ produced by measles favouring the growth of diphtheria bacilli. J. D. Rolleston.

Erosion of the common carotid in diphtheria (*'Inaugural Dissertation, München,'* 1909).—**O. Lukinger.**—A boy, aged $6\frac{1}{2}$ years, was admitted to hospital on November 5 with severe faucial diphtheria. He had been ill three days. There was much albumin in the urine. On November 6 a peculiar hæmorrhagic vesicular eruption appeared first on the skin and then on the buccal mucosa. No improvement followed two injections of antitoxin. On November 10 he had a severe attack of dyspnœa. Intubation produced relief, but tracheotomy had to be performed the next day. On the 14th the tube was left out, but had to be replaced the next day. On the 17th the tube was again removed. The wound was then gaping and fœtid pus was escaping from the trachea. About an hour after the removal of the tube a thick gush of blood poured out of the trachea and death took place in eight seconds. Necropsy: Erosion of the common carotid at its origin by an abscess cavity which communicated with the trachea. Deep ulceration of larynx, superficial ulceration of trachea, aspiration of blood and pus into lungs. Perforation of subpleural abscess of left upper lobe with circumscribed fibrino-purulent pleurisy. Subacute nephritis, œdema, and cloudy swelling of liver. Erosion of the neck-vessels has been recorded in scarlet fever, but never before in diphtheria. [The description of the case resembles one of scarlet fever rather than diphtheria. No mention is made of a bacteriological examination.—J. D. R.]

J. D. Rolleston.

Return cases of diphtheria and scarlet fever (*Hospitalstidende*, 1911, LIV, *p.* 441).—**Sörensen.**—7037 diphtheria patients discharged from Blegdom hospital during the period 1898–1908 yielded a return case percentage of 1·16. In the first quinquennium, when diphtheria was more prevalent in Copenhagen than subsequently, there were 54 return cases or 1·32 per cent. out of 4088 discharges; during the following six years there were 28 return cases or 0·95 per cent. The majority of the return cases were admitted four to twelve days after the discharge of the primary cases. Seventy-three out of 81 return cases had been free of diphtheria bacilli in the two successive cultures taken before their discharge. Among 8309 scarlet fever patients discharged in the period 1902–1909 the return case percentage was 3·7, or 305 cases, among whom the death-rate was 7 per cent. as contrasted with a mortality of 2·5 per cent. among the total cases. All the diphtheria return cases recovered. J. D. Rolleston.

The high death-rate from diphtheria in the United States (*'Med. Record,'* 1911, I, *p.* 568).—**E. C. Hill.**—The death-rate from diphtheria in New York State is four times greater than in Paris, and in New York City it is higher still. Thus from 1902 to 1909 it has ranged from 28·4 per cent. in 1905 to 39·2 per cent. in 1903 in New York State, and in New York City from 37·1 per cent. in 1908 to 57·0 in 1904, as compared with a mortality in Paris varying from 7 to 20 per cent. in the same period. A similar high death-rate from diphtheria prevails throughout the United States. The highest mortality in 1908 occurred at Washington, where it was 35·4 per

cent.; the lowest mortality in the same year was 11·34 per cent. at Vermont. The high death-rate is attributed to late and ineffective prophylactic measures, late diagnosis, lack of faith in antitoxin, and insufficient dosage in severe cases. Five illustrative cases are recorded. J. D. Rolleston.

Incidence of diphtheria in India (*'Indian Med. Gaz.,'* 1911, xlvi, *p.* 175).—**C. J. Fox**, judging from the number of positive findings in the examination of throat swabs sent to the Kasauli laboratory during the last five years, thinks that diphtheria is much commoner in India than is supposed, especially as the swabs were chiefly from severe cases and not from those which originated them. He records six cases, five of which were examples of membranous rhinitis and one of a mild angina. The mortality from diphtheria in India is much less than in Europe, where the disease is more virulent. J. D. Rolleston.

Scarlet fever in Philadelphia (*'Med. Record,'* 1910, ii, *p.* 1053).—**A. K. Sallom.**—During the past twelve years 32,317 cases of scarlet fever occurred in Philadelphia. The mortality ranged from year to year from slightly less than 3 per cent. to a little over 7 per cent. The average morbidity in January was rather high, falling in February, rising in March, and falling again in April. The highest number was reached in May, and the lowest in July. The morbidity then rose slowly at first and then more rapidly as the cold whether appeared. J. D. Rolleston.

Scarlatina and lactation (*'Arch. de méd. des Enf.,'* 1911, xiv, *p.* 124).—**Buffet-Delmas** records a case of severe though uncomplicated scarlatina in a nursing woman who was nevertheless able to carry on lactation uninterruptedly. The child, aged 1 month at the onset of his mother's attack, suffered no ill effects, and his weight week by week corresponded with that of a child suckled under the most favourable conditions.
J. D. Rolleston.

Scarlatinal thyroiditis (*'Monatsschr. f. Kinderheilk.,'* 1911, ix, *p.* 560).—**J. Bauer** records three cases occurring in very mild scarlet fever. (1) Boy, aged 10 years. Enlargement of left lobe of the thyroid appeared on the forty-sixth day, gradually subsided in the course of ten days, but was still palpable on his discharge from hospital on the sixty-sixth day. (2) Girl, aged 18 years. Enlargement of the thyroid, especially the right lobe, on the eleventh day. This slowly subsided, but was still perceptible on her discharge on the forty-third day. (3) Boy, aged 13 years. Enlargement and slight tenderness of the right lobe on the seventeenth day. In none of the cases were there any cutaneous redness, dyspnœa from pressure on the trachea, nor circulatory disturbance. J. D. Rolleston.

Albuminuria in scarlet fever and its relation to diphtheria (*'Thèses de Paris,'* 1910–11, *No.* 1).—**R. J. M. Pougaud.**—Late albuminuria in scarlet fever is often of diphtheritic origin, as is proved—(1) by the frequency of concomitant diphtheria, the clinical signs of which are confirmed by bacteriological examination; (2) by the frequency of diphtheria bacilli in the upper respiratory passages of scarlet fever patients without appreciable clinical signs; (3) by the effects of antitoxin treatment. Thus in a series of 128 scarlet fever cases treated with prophylactic injections, only two patients, or 1·56 per cent., developed albuminuria, as compared with eleven patients,

or 9·16 per cent., out of 120 not so treated. In the presence of existing albuminuria curative doses of antitoxin should be administered every four days. The late albuminuria of scarlet fever is not, however, always of diphtheritic origin, for it may occur in spite of prophylactic treatment and persist after repeated injections. The thesis is based on observations in Lesage's wards at the Hôpital Herold, and contains the histories of twenty-five cases, all but five of which are original. J. D. Rolleston.

The association of measles and scarlet fever (*'Paris Méd.,'* 1911, I, *p.* 191).—**V. Hutinel** finds that measles and scarlet fever can be associated in the following ways: (1) Measles may precede scarlet fever. Some authorities consider that the gravity of the latter is not thereby increased; others hold the opposite opinion. Hutinel inclines to the former view and quotes five cases in support of it, but at the same time he states there are exceptions, and instances two cases in which death rapidly occurred after the scarlet fever rash appeared. He finds this is most likely to occur if measles has been complicated by broncho-pneumonia. (2) Measles and scarlet fever appearing simultaneously. This association is not necessarily of serious omen. Broncho-pneumonia is rare in cases of this nature. (3) Measles following scarlet fever. Most clinicians admit that this association is serious, all the more so the nearer measles follows scarlet fever, but this view is not universal Hutinel has had four deaths in fifteen cases. The serious cases were nearly always those in which measles showed itself four to eight days after the scarlatinal eruption; if, however, a long interval occurs, the result is usually favourable.

F. R. B. Atkinson.

Epidemic of abnormal varicella (*'Thèses de Paris,'* 1910 11, No. 201).—**P. Le Roy** records an epidemic of thirteen cases in the crêche at Nanterre among children aged from four months to two years. In two the skin eruption was entirely papular, though one of the two showed a typical vesicle on the palate. In the other cases the eruption was chiefly papular, but the vesicles, though scanty, were quite characteristic. The other abnormalities observed were otitis media (1 case), abscess of little toe (1 case), and scarlatiniform rashes (2 cases). All the children recovered. The thesis contains the histories of twenty-four cases; thirteen are original, and eleven are examples of scarlatiniform rashes in varicella taken from the literature.

J. D. Rolleston.

Renal and articular complications of varicella (*'Semaine Méd.,'* 1911, XXXI, *p.* 140).—**Rosenblatt** and **I. Bienstock.**—a youth, aged 16 years, who three years previously had had severe scarlet fever complicated by nephritis, fell ill with varicella. The day following the appearance of the eruption the urine contained a large quantity of albumin, and on microscopic examination granular casts and red cells were found. The patient was somnolent and apathetic for ten days, during which his temperature ranged from 99·4° to 102° F. The albumin gradually diminished, and in eighteen days only a trace was left. The temperature then suddenly rose to 101·4° F, and there was marked swelling of the right radio-carpal joint. For three days the temperature ranged from 99° to 102° F. Shortly afterwards the right knee-joint became affected. Examination of the blood showed a well-marked leucocytosis, but no micro-organisms. Subsequent recovery was uneventful. J. D. Rolleston.

The contagiousness of whooping-cough (*'Gaz. des Hôp.,'* 1911, LXXXIV, *p.* 69.)—**Lesage** and **Collin**.—Every case of whooping-cough is accompanied by a rise in the specific gravity of the urine and by an increase in the uric acid, as well as by lymphocytosis and characteristic expectoration. The end of the contagious period corresponds clinically with the cessation of expectoration, a marked diminution in the number of the nocturnal paroxysms, and a return to normal of the blood and urine. The duration of the isolation period can thus be settled. There is no justification for regarding as a prolonged attack a morbid state which is characterised merely by the persistence of the whoop. In the absence of a definite relapse the persistence of the whoop beyond a month if the blood and urine are normal indicates that the cough has become a tic for which firm moral treatment is required. **M. Regnault** (*'Thèses de Paris,'* 1910–11, *No.* 238), in a thesis in which the above views are incorporated, records thirteen illustrative cases observed in Lesage's wards at the Hôpital Hérold.

J. D. ROLLESTON.

A case of Hymenolepis nana (*'Indian Med. Gaz.,'* 1910, XLV, *p.* 259).—**J. Davenport Jones** reports the case of this rare tape-worm in a European boy, aged 5 years. Treatment with filix mas in capsules resulted in the expulsion of a large number of worms, probably over one hundred. The worms were best seen and isolated by shaking portions of the fæces with water in a glass beaker held against a black background. In most of the specimens the heads were broken off, but the author found a perfect head, which proved to be a typical *Hymenolepis nana*. The worms are very small and easily overlooked, and are not expelled by the administration of santonin.

JAMES E. H. SAWYER (Birmingham).

Tape-worm in an infant aged 10 months (*'Brazil Medico,' August* 22, 1910, *p.* 313).—**Sarabia y Pardo** describes the infant as having been throughout suckled by the mother. It gained weight regularly till the fifth month, when it commenced to lose without any apparent cause, until a portion of the *Tænia* was found in the stools. This was found to be *Tænia solium*. After treatment by small repeated purgatives the infant soon commenced to recover. The parasite was probably conveyed in a drink of water on some occasion.

M. D. EDER.

Tapeworm in a baby (*'Arch. de Méd. des Enf.,'* 1911, XIV, *p.* 525).—**J. Comby** records a case of *Tænia saginata* in a female child, aged 9 months, who had been infected by the ingestion of raw beef-juice. Extract of male fern was prescribed and several segments were passed, but the head was retained until an emulsion of fresh pumpkin seeds had been given. The rarity of tapeworm in the first year of life is shown by the fact that this was the first case that Comby had met with in thirty years' practice.

J. D. ROLLESTON.

Numerous ascarides in several members of the same family (*'Jahrb. f. Kinderheilk.,'* 1911, LXXIII, *p.* 352).—**I. Péteri**.—In the course of a few months the first child evacuated 34, the second 175, the third 449, and the fourth 59 ascarides in the stools or vomit. The children had probably been infected by playing in a dirty courtyard. Their mental and physical development was not affected, and there was hardly any constitutional disturbance.

J. D. ROLLESTON.

Polyarthritis of obscure origin (*'Indian Med. Gaz.,'* 1910, XLV, *p.* 344).—**Drury** reports febrile polyarthritis in two native Indian children which was not influenced by salicylates. In one case Major L. Rogers cultivated from blood obtained from the joints a minute staphylococcus which grew in colonies resembling colonies of streptococcus. Vaccine treatment of this patient was at first followed by a slight improvement, but as soon as the dose of vaccine was increased the articular pain and the fever increased. The vaccine treatment was stopped. Eventually the patient, a boy, aged 12 years, improved very considerably on guaiac and passive movements.

H. D. ROLLESTON.

Chronic rheumatoid arthritis of childhood (*'Arch. of Pediat.,'* 1910, XXVII, p. 652; and *'Trans. Amer. Ped. Soc.,'* 1911, XXII, p. 72).—**H. Koplik** details cases, with general remarks on this conditions and its relation to other joint affections in children. He excludes all articular conditions which ordinarily complicate infectious diseases and gonorrhœa, and the results of syphilis and tuberculosis. He concludes that the condition is infective in origin, as shown by the mode of onset, the enlargement of the spleen, lymphatic glands and liver, the fever, and by the presence in some instances of leucocytosis and cutaneous hæmorrhages. He recognises that enlargement of the lymphatic glands is not absolutely constant, and that the enlargement of the glands, spleen, and liver is mainly an early phenomenon and may disappear. This glandular enlargement is much more marked than in adult rheumatoid arthritis. Emaciation and anæmia are characteristic, and exophthalmos may occur. He records in detail five cases—three in males and two in females—which were fully examined as regards the presence of syphilis and tuberculosis by tests which showed their absence. In four out of these five cases the lymphatic glands were enlarged and in one there was a leucocytosis of 20,000. The literature is quoted, and full credit given to Professor Still.

H. D. ROLLESTON.

The chronic polyarthritis of childhood (*'La Pediat.,'* 1911, XIX, *p.* 81).—**Prof. C. Cattaneo** contributes an original paper on this subject based on four cases under his care, derived from more than 15,000 patients in the first decade of life. The numerous designations given to this disease, such as chronic polyarthritis, nodular rheumatism, progressive ankylosing arthritis, Still's disease, etc., have in no way given any clear idea of it. In true chronic polyarthritis there is never complete resolution, although some improvement may occur, and this, according to the author, constitutes the fundamental nature of the disease. It is true that syphilitic chronic polyarthritis may be cured by specific remedies, while the other is not; but this is a therapeutic criterion, and not a difference in clinical aspect which distinguishes them. Still places in a class by itself a form, of obscure origin, in which, besides the articular lesions, there is enlargement of the glands and spleen. Chronic polyarthritis of toxic origin has been described under the name of "chronic thyroid rheumatism" by Pathon and others. Lévi and Rothschild, who have collected all that is known as regards its pathology, express the opinion that thyroid disturbance may have some pathological importance in chronic polyarthritis by altering the soil, by which it becomes possible for toxic causes to determine the chronic articular lesions. The author does not agree with Poncet's view that every case of primary chronic polyarthritis is a tuberculous rheumatism. The author's cases were aged 2, 6, 4, and 8 years, and are reported in full. In the majority of

the cases the articular condition was secondary to scarlatina or measles, indicating that some pyogenic infection was probably the cause. The pathological changes did not allow of any substantial differentiation between the various cases, whether primarily or secondarily chronic, except in rare instances in which tuberculous lesions were found. The author asks whether there exist types of chronic polyarthritis characterised by various modes of origin, clinically different in the condition of the articular lesions, complications, course, and termination. He considers they may be divided into two classes, one in which the first manifestations are in the small joints, and the other in which the large joints are affected from the beginning. In the first type the course is more rapid, terminating in ankylosis and deformity; in the other the course is milder, without great deformity, and with possibility of improvement, or even arrest of the morbid process. VINCENT DICKINSON.

Surgery.

Hereditary cranio-cleido-dysostosis (*'Lancet,'* 1910, II, *p.* 1466).—**Duncan C. L. Fitzwilliams** analyses sixty cases, two of which came under his personal notice, while the rest were all that could be collected from the literature. The hereditary nature of the disease is well shown, eight families supplying no less than thirty-one affected inviduals. The facial and cranial changes are discussed, and considered to be due to the same cause that produces the characteristic features in hydrocephalus, namely that the base of the skull fails to develop with the increased growth of the brain and is accommodated by the opening of the vertex. The musculature of the altered shoulder-girdle is given at length. The state of the clavicle is analysed, and shows that while the sternal portion is hardly ever absent the acromial portion is almost invariably lacking. The ligamentous prolongations, which replace the acromial portion in the majority of cases, do not extend to the acromion but go to the coracoid process. These facts throw considerable light on the development and morphology of the clavicle. This bone has long been suspected of being the result of the fusion of two separate elements, of which one is preformed in cartilage and the other in membrane. The disease hereditary cranio-cleido-dysostosis retards and prevents the development of bones preformed in membrane. The probable fate of the coracoid and precoracoid bars of cartilage in the promitive shoulder-girdle is then discussed. The writer shows that the inner two thirds of the clavicle, the coraco-clavicular ligament, and the base of the coracoid process itself are probably all part of the same bar, while the tip of the coracoid process and the bicornuate ligament are probably parts of a second bar. In one of the author's cases the first of these bars was intact and stretched from the sternum to the glenoid cavity. The dissections performed in this disease and the method of ossification of the coracoid process both tend to support the idea that the inner two thirds of the clavicle, the costo-coracoid ligament, and the base of the coracoid process are the representatives of the true coracoid, while the bicornuate portion of the costo-coracoid membrane and the tip of the coracoid process represent the old precoracoid. The positions of the two bars are slightly modified by muscular attachments, while the acromial third of the clavicle is of a more recent ancestry, membranous in origin, and possibly related to the dermal plate of lower vertebrates. Other possibilities are discussed, but seem to be less likely than the conclusions mentioned. As to the cause of the disease nothing definite can be said; its

relations to achondroplasia are mentioned, and the suggestion thrown out that both may be due to a lack of some chemical substance or enzyme which is needed for sound ossification. AUTHOR'S ABSTRACT.

Case of femoral dysostosis with obesity (*'La Clin. Infant.,'* 1911, IX, *p.* 230).—**MM. Variot** and **Chatelin** showed this case at the Soc. Méd. des Hôpit. The girl was aged 15 years and measured 1·28 m. (average 1·54 m.) and weighed 38 700 k. (average 26 k.). The diminution of height was due entirely to the shortness of the femora, which were also curved. There was an over-development of fat, chiefly on the chest, stomach, and thighs. Breasts small; menstruation absent. Intelligence normal. Radioscopy showed slight enlargement of the sella turcica, but no lesions characteristic of achondroplasia. The arms were normal. There was consolidation of the femoral epiphyses, which usually does not take place till the age of twenty. VINCENT DICKINSON.

A case of fragilitas ossium (*'Zentralbl. f. Kinderheilk.,'* 1910, XV, *p.* 504).—**A. H. Meyer** exhibited at the Danish Pædiatric Society a child, aged 11 months, suffering from rickets and severe colitis who had been fed on a mixture of water-gruel and pasteurised milk. Fractures were present in all the diaphyses of the extremities. The X rays showed atrophy of the bones, consisting in deficiency or maldevelopment of the compact layer and feeble development of the spongy layer, the trabeculæ of which were scanty. There was no sign of Barlow's disease. J. D. ROLLESTON.

Idiopathic osteopsathyrosis (*'Jahrb. f. Kinderheilk.,'* 1911, LXXIII, *p.* 545).—**S. Miura** records a case in a Japanese girl, aged 10 years. There was no family history of brittle bones. Since her first year she had had nineteen fractures, the deformities resulting from which rendered standing and walking impossible. Rickets, osteo-malacia and Barlow's disease could be excluded. The X rays showed considerable thinning of the compact layer, rarefaction of the spongy layer, and normal epiphyses. J. D. ROLLESTON.

Fracture of the scapula in a child (*'Paris Méd.,'* 1911, I, *p.* 451). — **F. Jacoulet** describes a case of this rare condition in a boy, aged 13 years, resulting from a fall from a height of from five to six metres. The fracture crossed the sub-spinous fossa. The author quotes Végas and Joye, who found, out of 500 fractures, only one of the scapula, in a child aged 4 years. The youngest case seems to be Hill's (six months). The fractures are always caused by direct violence. The prognosis is favourable. The treatment consists in the application of a bandage, keeping the parts immobile, with the arm slightly backwards. F. R. B. ATKINSON.

Treatment.

The intra-venous injection of antitoxin in diphtheria (*'Die Therapie der Gegenwart,'* 1910, LI, *p.* 346).—**H. Tachau.**—One hundred cases were treated by this method in the Frankfort Municipal Hospital in the course of the year. In 78 the diagnosis of diphtheria was confirmed by bacteriological examination. The usual technique was employed. Special care was taken to inject slowly. Exposure of the veins was not undertaken, and the method was therefore not adopted in small children

in whom direct puncture of the vein was impracticable; 3000–4500 units were given in the mild, and 6000–9000 units in the severe cases. As a rule only one injection was given; only a few very severe cases received a second injection on the following day. Nine of the 78 cases died—a mortality of 12·8 per cent., as compared with one of 14 per cent. among 170 cases treated subcutaneously. These figures do not speak in favour of the intra-venous method when it is considered that small children in whom diphtheria very frequently takes a more unfavourable course were almost all treated subcutaneously. The cause of death was either toxæmia or circulatory and renal complications, which intra-venous injections had therefore no power to prevent. In mild cases the intra-venous method did not tend to shorten the course of the disease, the clearing away of the membrane and the disappearance of bacilli from the throat being no more rapid than in cases treated subcutaneously. That the intra-venous method was not unattended by risk was shown not only by a rise of temperature following injection in about half the cases, but also by alarming symptoms of collapse occurring in three cases, though these had received no previous injection, so that anaphylaxis could be excluded. In one case an erythema developed which was regarded as a vaso-motor disturbance associated with the collapse rather than as a serum eruption. Tachau concludes that intra-venous injection has no therapeutical advantages over subcutaneous injection, but on the contrary has some serious drawbacks. He has therefore abandoned the method, especially as it is more complicated than the ordinary one.

J. D. Rolleston.

Contribution to the study of human anaphylaxis (*'Riv. di Clin. Pediat.,'* 1910, vii, *p.* 915).—**U. Calcaterra** describes three cases of diphtheria treated by serum, first by subcutaneous and then by intra-spinal or intra-venous injections. One died. In all, the phenomena of anaphylaxis were observed, and the author concludes that intra-spinal injections should be used with the greatest caution, especially in diphtheria, not only when the patients have been treated by serum some time before, but even two or three days previously.

Vincent Dickinson.

Intra-spinal serotherapy in diphtheria (*'Riv. di Clin. Pediat.,'* 1910, vii, *p.* 981).—**C. Francioni** confirms some of Calcaterra's views (*ibid., p.* 915). As the intra-spinal method is directly efficacious in preventing toxic lesions of the nervous system, he is of opinion that in studying statistics account should not merely be taken of recoveries or deaths, but in cases which survive attention should especially be given to the more or less pronounced character of the late paralytic manifestations and an estimate formed from them of the efficacy of the treatment. The task of curing the degenerative visceral lesions, which are those most frequently responsible for the rapidly fatal issue in the more toxic forms, must be still reserved for hypodermic, intra-muscular, or better, intra-venous sero-therapy. According to the author, the only effect of intra-spinal injections is to render less intense and dangerous the outbreak of late paralytic manifestations, or at least those of them which are attributable to toxic lesions of the nervous system. Hence the intra-spinal method is only required in the most serious cases of unfavourable prognosis in which we can foresee the disappearance of grave paralytic conditions at a later period.

Vincent Dickinson.

The results obtained in scarlet fever with "scarlatin Marpmann" (*'Allg. Wien. med. Zeit.,'* 1910, LV, *p.* 448).—**Hartung** has collected from 312 medical men 1915 cases where the remedy has been used, with a mortality of 47, or 2·4 per cent. This contrasts favourably with the 5 or 6 per cent.—usual mortality. Out of the 312 doctors only five were adverse, but there was only one case of any untoward effect where urticaria set in. There was general agreement that the earlier the use of the remedy the more clearly marked was its value. The most noticeable effects were the reduction of the temperature, the strengthening of the pulse and quieting of the patient. Prophylactically the antitoxin was used for 2126 children where infection was feared; out of these 68 children were attacked by scarlet fever—among these 68 were two deaths. These figures must be regarded as extremely favourable to the more extended use of the preparation both as a preventative in epidemics and as treatment in cases of illness. M. D. Eder.

Treatment of whooping-cough in infants (*'Jahrb. f. Kinderheilk.,* 1911, LXXIII, *p.* 728).—**Mehnert**, since 1906, has found that vaccination has a curative effect upon pertussis in hitherto unvaccinated infants aged from three to nine months. During the development of the pustules the cough becomes less severe and disappears entirely within a fortnight (*cf.* British Journal of Children's Diseases, 1908, V, *p.* 218). J. D. Rolleston.

Treatment of normal whooping-cough (*'Paris Méd.,'* 1911, I, *p.* 563).—**P. Nobécourt** describes some of the remedies not usually known for this disease. Weill and Martin found good results from the inhalation of quinoleine, a substance extracted from coal-tar or prepared synthetically. Ten to thirty drops are added to 100 c.cm. of boiling water and inhaled three or four times a day up to twenty minutes. Internally a saturated solution of fluoroform is well spoken of by Tissier. Two gr. of 8 per cent. fluoroform is given three times a day in the following doses: Up to one year, 10 to 15 drops; from one to three years, 15 to 20 drops; over three years, 20 to 30 drops, and in addition after each attack of coughing as many drops as months or years of the child's age: 60 to 80 drops can be given *per diem* in the newly-born up to 150 to one year and to 20 grm. to children over three. Anti-diphtheritic serum, or 0·10 gr. of anti-tetanic serum, have also proved efficacious. The author devotes the remainder of the article to a consideration of the usually employed remedies and sums up the treatment as follows: Inhalations of water medicated with various substances, as quinoleine, etc., internally, belladonna, quinine, or antipyrin in increasing doses. If the fits of coughing are violent, inhalations of oxygen, chloroform or ether during the attacks, and if very severe, morphia injections or chloral. Hygienic measures are essential.

F. R. B. Atkinson.

Reviews.

The Disorders of Post-natal Growth and Development. By Hastings Gilford, F.R.C.S. London: Adlard and Son, 1911. Royal octavo; pp. xxii + 727, with 65 figures. Price 15*s.* net.

Mr. Hastings Gilford is already widely known in connection with his writings on ateleiosis and progeria, and naturally, therefore, his ideas con-

cerning the disorders of growth and development, as expressed in the present volume, will excite great interest amongst all those in any way interested in the subject. It is at least no exaggeration to say that every medical man should be interested in the subject, for Mr. Gilford has searched all the various departments of pathology and biology to illuminate it. His comparisons are always striking and his generalisations are far-reaching. For instance (p. 107), he points out that the major variation of albinism in an English fox would constitute to all intents and purposes a disease, for it would render the animal conspicuous, attract the attention of the hounds, give warning to the animals upon which it preys, and therefore lead, just as surely as heart disease, etc., to defective nutrition and an early death. How clearly expressed (p. 28) is the comparison of the cells and organs of the human body to unicellular and more complex organisms living together for mutual advantages. "A kidney may, in short, be regarded as comparable with an organism of high development, one of a series which is shut off from the general somatic system by abrupt boundaries, working for and owing allegiance to the whole body, coming into touch with it when the general good is concerned, but in other respects living independently." Another instructive comparison is made on p. 34, namely, that between the state of pregnancy and the normal pre-menstrual state. In an early part of the book the toxins produced by pathogenic microbes within the body, which give rise to enteric fever and other zymotic diseases, are compared to alcohol, strychnine, and other toxins and drugs produced by non-parasitic plants. This latter group both cause disease and are used for the cure of disease. Might one not go further and compare them to antitoxins and other substances (produced by cells of the animal body), which help the body to resist disease and maintain its normal functions? Is it not possible that antitoxins of this nature may sometimes have a harmful as well as a salutary effect? It has, we believe, been suggested that the degenerative changes of tabes dorsalis and general paralysis may be due to the prolonged action of syphilitic antitoxins.

Mr. Gilford's subject, as discussed by him, is such a vast one that in a short notice like this it is quite impossible to give an adequate idea of the scope of the work. Those who jump to the conclusion that the book deals exclusively or even chiefly with ateleiosis and progeria, for which the author is already so well known, will find themselves greatly mistaken. Every medical practitioner should read the book for himself—not read hurriedly through it, but read in it at spare moments, and thus gradually finish reading it at his leisure, in which case it will certainly become for him a source of much pleasure as well as of instruction. The author might, perhaps with advantage, have devoted a little more space than he has done to the modern observations on the relation of the adrenal bodies and the hypophysis cerebri to disorders of general growth and development other than acromegaly and gigantism. F. P. W.

THE MEDICAL DISEASES OF CHILDREN. By REGINALD MILLER, M.D., M.R.C.P. Bristol: John Wright & Sons, Ltd., 1911. Pp. 541. Price 12s. 6d. net.

DR. R. MILLER has written a book which deserves to become popular with the profession, inasmuch as it is a very complete and lucid manual of the medical diseases of children. The whole subject is treated in a very able manner, and the fact that the author has drawn from his own experience

and from that of the most modern writers ensures that the reader will find the current ideas embodied in the work. This alone should render the book useful to students and practitioners alike.

Dr. Miller writes in an incisive, though not too dogmatic, style. His descriptions are convincing and avoid being prolix, while the hints which he gives as to treatment and technique will be found not the least useful part of the book. The value of the text is enhanced by a large number of photographs, which are in every way admirable, and serve to impress the conditions depicted the more vividly upon the mind.

As in most works on children the book opens with sections on the Examination of children and on Development and Feeding, which are clear and simple. Then follow the various diseases under their appropriate headings. Where there is so much that is good it is difficult to single out any part for special mention, but perhaps the section on Infective Diseases is the best of all. In this the author deals with pneumococcal, tuberculous and rheumatic infections, inherited syphilis, diseases caused by the meningococcus and gonococcus, acute polioencephalo-myelitis and the infectious fevers, to mention only some of the more important. At the end are appendices on Dietetic and Therapeutic Measures and on Societies and Institutions aiding invalid Children, which form handy and reliable sources of reference.

The get-up of the book is in every way excellent. It is printed in clear type and on glazed paper; the latter, however, has the disadvantage of making the book somewhat heavy. The photographs, to which reference has been already made, are a prominent feature of the work, and great skill has been shown in their reproduction.

Altogether the book is one which we can thoroughly recommend.

T. R. W.

La Poliomyélite Epidémique. By Dr. George Schreiber. Paris: G. Steinheil, 1911. Price 10 francs.

This work is divided into two parts: the first deals with the disease described by Heine and Medin, and its relation to the "medullo-virus" of Landsteiner and Popper; the second with meningo-myelitis and meningitis in its relation to the "medullo-virus" of Landsteiner and Popper.

The first chapter of Part I discusses the use of the name "poliomyelitis." The author considers the name should be retained and regarded as a syndrome charcterised by the production of paralysis, due to an affection not exclusively but predominantly of the anterior horns and grey matter, induced most frequently by the "medullo-virus" of Landsteiner and Popper—an agent capable of attacking isolated individuals, but causing more often an epidemic, sometimes widespread, sometimes limited, and affecting different portions of the nervous system, giving rise to various forms which are known under the title of Heine-Medin's disease. The work deals with the history of the disease, the history of the pathological anatomy, the history of the epidemiology, the history of the clinical manifestations, and the history of the bacteriology of the disease.

A chapter is devoted to the experimental work based on the observations of Landsteiner and Popper, Flexner and Lewis, Leiner and Wiesner, and Levaditi. The clinical forms of the disease in the monkey are described, and their similarity to those in man demonstrated

The nature of the virus is then considered. The virus is shown to be filtrable, will resist glycerination for months, and desiccation for fifteen days. The virus is killed by exposure to temperatures of 40–50° F. for half an hour,

but resists cold of — 8° C. The organism cannot be cultured, is destroyed by various chemicals *in vitro*, and urotropin seems to exercise a destructive action on the virus *in vivo*.

Certain forms of monkey are susceptible to infection, but guinea-pigs, mice, rats, dogs, cats, sheep, pigs, goats, horses, fowls, and pigeons are insusceptible. Krause and Meinicke have produced paralysis in rabbits, but others have failed to do so in this animal.

The virus can be introduced into the system by the peritoneum, the brain, subcutaneously, intra-venously, by the digestive tract and the respiratory passages if injured. It is striking that no case of contagion took place in monkeys even when in the closest contact, and this is attributed to the integrity of the mucous membrane.

It is shown that the virus tends to reach the central nervous system by the path of the nerves, and that if a peripheral nerve is infected and then the nerve divided infection does not occur.

The elimination of the virus is probably by the nasal mucous membrane. The saliva, the urine, the bile and the fæces cannot be shown to contain the virus.

It is shown that immunity is easily produced in the monkey.

Chapters on the pathological anatomy and ætiology follow. With regard to the ætiology it is mentioned that the disease more frequently attacks bootmakers' children than those of any other occupation.

A list of cases in which two or more members of a family or household were affected is given. Further chapters deal with the clinical forms, the diagnosis, and treatment of the disease.

Part II discusses the relation of meningitis to cases of poliomyelitis which present meningeal symptoms.

The conclusion arrived at is that the "medullo-virus" may give rise to all the symptoms of a meningitis, and need not be accompanied by any paralysis.

The book is an excellent compilation of all the early and recent studies on poliomyelitis, but no short review such as the present can do justice to it.

The work is well put together, and the bibliography so arranged that it is easy to find the references to any given portion of the subject on which it is desired to obtain information. F. E. B.

ARCHIVOS BRASILEIROS DE MEDICINA. Redaçcao: R. Gonçalva Dias 26, Rio de Janiero.

WE have received the first four numbers of this journal, the excellence of which should secure it a prominent position among medical periodicals. The editors are Prof. Moreira, of Bahia, and Prof. Austregesilo, of Rio. In addition to six ordinary numbers supplementary issues are to appear every other month. The first number contains a paper by Figueira on infantile scurvy, and one by Alvaro Ramos on Madelung's deformity, to which is appended an extensive bibliography, which will be of value to readers not familiar with Portuguese, for whose benefit also each original article has been summarised in French or German.

Among the abstracts from current literature in the first and third numbers we are pleased to find that several pages have been devoted to children's diseases, and that this JOURNAL has been laid under contribution. The second number contains an account of a meeting of the Brazilian Pædiatric Society, in which alimentary fever, and cases of Pick's disease, meningomyelitis and *B. coli* pneumonia were discussed. J. D. R.